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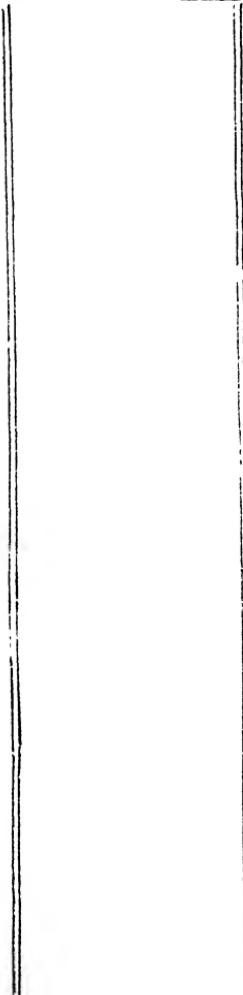
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POTATO CRINKLE, VAR. IRISH CHIEFTAIN.
This is a composite virus disease (see p. 391). (After Murphy.)

[Frontispiece.

A TEXTBOOK OF PLANT VIRUS DISEASES

By

KENNETH M. SMITH

D.Sc.(Manchester), Ph.D.(Cambridge)

*Potato Virus Research Station School of Agriculture,
and also the Motteno Institute, Cambridge*

WITH 101 ILLUSTRATIONS



LONDON
J. & A. CHURCHILL LTD.
104 GLOUCESTER PLACE
PORTMAN SQUARE

1937

“Until the phenomena of any branch of knowledge have
been submitted to measurement and number it cannot
assume the status and dignity of a science.”

FRANCIS GALTON.

PREFACE

To prepare a textbook on plant virus diseases, it is essential to have some scheme of classification of the viruses to enable the subject to be presented in a coherent form. In an effort to arrive at the most satisfactory solution of this problem, three alternative schemes for the book were outlined. These schemes were as follows : In the first, the subject was classified according to the *diseases* caused by the different viruses, and the whole was arranged in the order of the plant families. In the second, the *viruses* themselves were classified and numbered right through from "Plant Virus 1" to whatever the final number should be, without reference to a host grouping. The third scheme was a modification of that suggested by James Johnson of Wisconsin, and was a compromise between the first two, *i.e.*, the *virus* was classified, but it was grouped according to its first-described or most important host plant. The book was commenced under Scheme I, and was partly written before it was realised that a classification of the diseases was impracticable because it necessitated so much duplication and cross-referring. The book was then re-drafted in accordance with the second scheme which, although the most logical, proved unsatisfactory for the following reasons. By numbering the viruses right through, no opportunity is allowed for the expansion of the system, so that any new virus discovered after the inauguration of the scheme would be widely separated numerically from other viruses of its particular group. This disadvantage is most apparent in regard to the group of potato viruses. Another drawback to this scheme is the difficulty of remembering a virus by a number alone, and this fact would not make the task of teaching the subject any easier. It is also peculiarly liable to clerical errors. Thus, by a process of elimination, the third method of classification was selected. It is based on the tentative scheme suggested by James Johnson, but the generic Latin name of the host plant has been used instead of the English popular name, partly for the sake of consistency because many host plants have either no popular name or else several names which differ in different countries, and partly

because of the wider international application of the generic name. The writer's conception of the identification of the viruses is not the same as that of James Johnson, but the latter's designation, when known, is given in each case in the list of synonyms.

The classification adopted in this book then, consists of a grouping together of all those viruses which are chiefly associated with a particular host plant, and the viruses are then numbered 1, 2, 3, etc. The book follows a common plan throughout ; the virus is first dealt with ; its properties, mode of transmission, etc., are given, and then the diseases it causes, arranged according to the plant families, are described. The viruses as a whole are placed in the order of their plant hosts, following Hutchinson's system of classification. The insect vectors are described in some detail in a separate section of the book.

In order to lessen any disadvantage to the practical man which might arise from the scheme of classification adopted, an Appendix has been prepared in which all the more common virus disease symptoms are listed together with the virus which causes them. The page reference is appended in each case so that further information is easily accessible.

While this system is admittedly a compromise it seems to the writer to be the best method, at present available, of presenting the subject. No account is given of the technique and methods of study of the plant viruses because this aspect of the subject has been dealt with already in another volume in the Recent Advances series.

It is hoped that the book will lessen the confusion now existing in regard to the study of plant virus diseases, and that it will help to give the subject its rightful status as a distinct science.

Grateful acknowledgment is due to plant virus workers in many different parts of the world for their generous assistance and co-operation in the preparation of this textbook. The writer is particularly indebted to the following for reading and criticising those portions of the work in which they are specialists, and for allowing access to unpublished data : Mr. R. V. Harris, Professor Paul Murphy, Dr. W. H. Pierce, Dr. C. M. Tompkins, and Dr. W. J. Zaumeyer. Dr. Lee M. Hutchins kindly supplied the account of peach mosaic. Acknowledgment is also due to Mr. W. E. China and Mr. F. Laing for information in regard to some of the insect vectors.

Many of the original illustrations are from photographs taken by Mr. J. P. Doncaster, who also helped materially in the prepara-

tion of the manuscript for press. Dr. R. W. G. Dennis kindly assisted in the proof-reading, while Mr. F. A. Buttress was most helpful in the matter of references to relevant literature.

The writer is much indebted to the editor of the *Bulletin of Entomological Research* for the loan of the blocks of Figs. 15, 79, 83; to the editor of the *Gardener's Chronicle* for the blocks of Figs. 2A, 3A, 8; to the editor of the *Journal of the Royal Horticultural Society* and of the *Lily Yearbook* for the blocks of Figs. 38, 48, 51, 53 and 54, and of Fig. 13 respectively; to the editor of *Scientific Horticulture* for the block of Fig. 73A; to Professor Paul Murphy and the Royal Dublin Society for the blocks of Figs. 62, 63B and 65; to Dr. Hatton and the editors of the *Journal of Pomology* for the blocks of Figs. 16 and 17; to the editor of *Parasitology* for the block of Fig. 42; and to the editor of the *Annals of Applied Biology* and the Cambridge University Press for the blocks of Figs. 14, 37, 40, 47, 52, 55, 56, 73A and B, 75, 77, 96, 99 and 100. The Royal Society supplied electros of Figs. 60B, 64C, 68C and D.

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KENNETH M. SMITH.

CAMBRIDGE.

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TEXT BOOK OF PLANT VIRUS DISEASES

CHAPTER I

Delphinium Viruses 1-2; *Paeonia* Virus 1; *Anemone* Virus 1;
Brassica Viruses 1-4; *Matthiola* Virus 1; *Beta* Viruses 1-5;
Pelargonium Virus 1; *Passiflora* Virus 1; *Cucumis* Viruses 1, 2
and 2A.

DELPHINIUM VIRUS 1. Burnett

Synonyms. *Delphinium Stunt Disease Virus*, Burnett, 1934;
Delphinium Witch's Broom Disease Virus.

The Virus and its Transmission. The virus is sap-inoculable; it, apparently, is not carried in the seed. The natural mode of transmission is not known. There is no information on the physical or other properties of the virus. The two viruses affecting delphinium, here described, bear some resemblance to *Cucumis Virus 1*, and may eventually prove to be strains of this virus.

Differential Hosts

Nicotiana tabacum var. Connecticut Havana. With the first transfer from delphinium to tobacco, symptoms may not appear until about the fifteenth to twentieth day, but sub-inoculation to tobacco frequently produces symptoms in four to six days. These symptoms are variable, sometimes showing as a white necrotic etching, with ring-and-line patterns of variable forms, while in other cases the first recognisable symptom is a vein-clearing. Some plants never produce the ring-and-line pattern, but exhibit a vein-clearing that later changes to a blotchy, irregular, usually interveinal mottle which may coalesce to form extensive blotchy chlorotic areas. In more mature plants these blotches may involve the major part of the older leaves. In addition the tobacco leaves usually are somewhat ruffled, producing a wavy and crinkled appearance. Some leaves may appear normal, but subsequently produce a faint mottle which may or may not develop into the blotchy type. The necrotic ring-and-line patterns are confined

2 DISEASES CAUSED BY DELPHINIUM VIRUS 1

to the first few leaves, while the older plants seldom show evidence of this symptom, but rather exhibit the blotchy type of foliage. Dwarfing of the whole plant is also usually evident (8).

Stellaria media. Chickweed. When chickweed is inoculated with the virus it becomes dwarfed and produces a mottle with chlorotic interveinal tissue. The veins usually retain their green colour, but, in some cases, they also become chlorotic. The apical leaves may become completely folded over, with the midrib as the central point of the fold.

It is worth noticing that *Datura Stramonium*, which is a differential host for so many viruses, including *Cucumis Virus 1*, is apparently resistant to infection with *Delphinium Virus 1*.

Diseases Caused by Delphinium Virus 1

Ranunculaceæ

Delphinium sp. Perennial delphinium. *Stunt disease.* The symptoms of the stunt disease are rather variable, but dwarfing of the plant appears to be characteristic of infection. This symptom is progressive and leads to the eventual death of the plant. Infected plants grown in the open are slow in starting spring growth as compared with healthy plants. Extreme dwarfing together with chlorotic, mottled or savoyed foliage is characteristic of infection in some plants, the chlorosis being usually confined to the leaf margins. An outstanding symptom is the development of brown or tarry black necroses either on the leaves or on the stems. Occasionally these black necroses may girdle the stem or petiole causing the death of the shoots. Later in the summer there may be excessive proliferation of the flowering stalks, which produce a bunched or witch's broom appearance. The flowering parts fail to develop normally and produce a characteristic leafy proliferation with varying degrees of transformation from slightly greenish flowers to pale green leafy structures.

The chief symptoms of the disease on delphinium may be briefly enumerated as follows :—

- (1) Retarded growth with varying degrees of stunting or dwarfing.
- (2) Chlorosis, savoying and cupping of the leaves.
- (3) Appearance of dark brown or black necrotic areas on leaf blades, petioles and stems.
- (4) Multiplication of weak and slender shoots in the more advanced stages of the disease.
- (5) Lodging of the tops.

(6) Premature yellowing of foliage and death of shoots at the time of, or just after, flowering.

(7) Increased proliferation of the flowering stalks with greening of the flower parts.

Compositæ

Zinnia elegans. Jacq. There is no mottle apparent on infected zinnias and the more evident symptoms consist of dwarfing, excessive crinkling and rigidity of foliage.

Lactuca scariola. The symptoms on prickly lettuce consist of dwarfing, a definite curling and folding of the leaf, and a blotchy mottle which is produced by numerous small, white necrotic areas.

Cucurbitaceæ

Cucumis sativus. On cucumber foliage the more pronounced symptoms consist of a mosaic mottle and rigidity of the foliage similar to that produced by *Cucumis Virus 1* (cucumber mosaic virus). Often there is also a sharp downward or an upward bending of the distal portion of the leaf. This downward or upward turning may be slight or it may form an angle of 90 degrees. The fruit does not seem to show any very clear symptoms.

Solanaceæ

Lycopersicum esculentum. Tomato. Twelve to fifteen days after inoculation tomato plants are usually somewhat dwarfed. The first symptom, other than dwarfing, is a definite vein-clearing, evident between fifteen and twenty days after inoculation. This is followed in a few days by a definite downward rolling of the leaflet margins and a hooking down of the ends of the leaflets as well as the apical part of the leaf. The leaflets often become deeply lobed or cut. The foliage at this stage shows a faint but perceptible mottle which intensifies with the age of the plant. The interveinal tissue becomes lighter coloured in contrast to the darker areas bordering the veins. As this mottling increases there appear irregular, large, blotchy areas on the leaves similar to those described on tobacco. Plants usually exhibit a pale green colour, and there is a slight to moderate savoying of the leaves. The fruit is apparently not affected. These symptoms are soon followed by further distortion of the leaves, in which they become curled in corkscrew fashion, turning on their axis as much as 180 degrees or

more. This curling also occurs on the main stem or on lateral branches (8).

Nicandra physaloides. Apple of Peru. The first evident symptom, appearing four to five days after inoculation, consists of water-soaked areas of variable size. These soon coalesce, resulting in large, irregular, brown necrotic lesions that may involve the entire leaf and cause it to die and fall from the plant with the possible resultant death of young plants. In any event the plant is materially dwarfed; but, if not killed, it will make a slow growth. The new foliage often may fail to produce evident symptoms, but the virus is systemic, since it can be recovered from the foliage that fails to exhibit symptoms.

Solanum nigrum. Black nightshade. *Delphinium Virus 1*, produces rather similar symptoms on *S. nigrum* to those produced by *Nicotiana Virus 1* (tobacco mosaic virus) on the same plants. The most common symptom is a mottling of light and dark green areas. There is no appreciable savoying of the foliage but a conspicuous, irregular blotchy type of mottle. *S. nigrum* appears to be less susceptible to *Delphinium Virus 1* than tobacco or tomato.

The following species are susceptible to infection, but show few or no symptoms: *Marrubium vulgare*, horehound; *Anthemis cotula*, dog fennel; *Capsella bursa-pastoris*, shepherd's purse; *Petunia hybrida*, petunia.

Geographical Distribution. Apparently quite general in the United States of America, the disease has been known in the State of Washington for about fifteen years. What is probably the same disease has been briefly described from Idaho by Hungerford (30) and from Indiana by Gardner (23). No records of the disease from other countries are known at present.

Control. Careful roguing and destruction of infected plants is an important measure. Special attention should be given to seedlings, as the early elimination of the disease will reduce losses to older plantings. Any vegetative propagation must be made from plants known to be free of the virus (8, 26).

DELPHINIUM VIRUS 2. Valleau

The Virus and its Transmission. The virus is sap-inoculable, but the insect vectors are not known. It is possible that the virus is transmitted by the seed, but this has not been definitely determined. Little is known of the physical properties of the virus

except that it has been proved unable to withstand desiccation. Further investigation may show this virus to be a strain of *Cucumis Virus 1* (cucumber mosaic virus).

Diseases Caused by *Delphinium* Virus 2

Ranunculaceæ

Delphinium sp. Perennial delphinium. There are chlorotic ring patterns on individual lobes and sometimes patterns which extend into each of the lobes of a leaf, following more or less closely the primary veins. The patterns may or may not be present as the plants start growth in the spring, but are likely to become more prominent as the leaves become older. After blossoming, a few or all of the leaves may bleach to a light greenish-yellow. Whether the disease is a cause of death after blooming has not been determined. From a limited number of observations it appears that the extensive chlorosis occurring at this time may be a factor in the failure to produce a healthy normal growth after death of the flowering stalks. The virus is occasionally carried without symptoms in delphinium.

Host Range. The host range includes plants in three families, Ranunculaceæ, Solanaceæ and Cucurbitaceæ. Of these the tobacco plant appears to be an important natural host.

Geographical Distribution. The disease is prevalent in garden varieties of perennial delphiniums in the vicinity of Lexington, Kentucky, Yonkers, New York and St. Paul, Minnesota. There appear to be no European records of the disease (67).

PÆONIA VIRUS 1. Dufrenoy

Synonym. *Peony ringspot virus*, Green, 1935.

The Virus and its Transmission. According to Dufrenoy (21) the virus is transmissible to tobacco, but only if inoculation is made from the chlorotic spots. The disease is prevalent in France in the region of Bordeaux and Brive, and it has been observed by Green in England (25).

Disease Caused by *Pæonia* Virus 1

Ranunculaceæ

Pæonia sp. *Peony ringspot*. Symptoms take the form of a marked yellow mosaic consisting of irregularly shaped patches of yellow on the leaves, occasionally accompanied by small necrotic spots. Frequently the yellow mottle is in the form of chlorotic

rings. Where an affected area meets a lateral vein the development of chlorosis is inhibited and the ring in consequence is incomplete (21).

ANEMONE VIRUS 1. Klebahn

Synonym. *Anemone alloiophylly virus*, Klebahn, 1926.

The Virus and its Transmission. The virus of alloiophylly is filterable and sap-transmissible, but there is no evidence of insect transmission. Klebahn has come to the tentative conclusion that the virus can remain viable in the soil or in decaying remnants of infected plants on the surface of the ground. He has obtained positive results in the inoculation of anemones with an extract of the soil adhering to the rhizomes of infected plants, as well as with the powder from dried leaves.

There is little known of the properties of this virus, and the disease seems only to have been described from Germany (33, 34, 35).

Diseases Caused by Anemone Virus 1

Ranunculaceæ

Anemone nemorosa. *Alloiphylly*. Affected plants show a strikingly spreading habit and the leaves are frequently much thickened and misshapen with abnormally shallow indentations. The stem and leaf stalks are almost always more or less noticeably thickened. The flowers are mostly suppressed, those that do occasionally develop being deformed. Certain anatomical modifications correspond with these symptoms (see Fig. 6, C).

Anemone ranunculoides and *A. trifolia* are also susceptible to this disease.

BRASSICA VIRUS 1. Smith

Considerable uncertainty still exists concerning the number and identity of the viruses which attack cruciferous plants. The following arrangement of these viruses is based partly upon the writer's own studies and partly on information kindly supplied by Dr. C. M. Tompkins from his unpublished work. The arrangement of the viruses, however, is solely the writer's responsibility. For the present, five viruses are classified and four others are briefly mentioned, there not being sufficient information to justify the classification of the latter. It may well be that further research will entail modification of this arrangement.

Synonyms. *Wallflower Mosaic Virus*, Smith, 1935; *Cabbage Ringspot Virus*, Smith, 1935; probably *Cabbage Blackspot Virus*, Tompkins, 1935.

The Virus and its Transmission. The virus is sap-inoculable and the insect vector is the aphid, *Myzus persicæ* Sulz. (see p. 538). There is no evidence that the virus is seed-transmitted. In the case of wallflowers the writer has found that seed from infected plants with "broken" flowers gives rise to normal plants with self-coloured flowers (62, 63).

Differential Hosts

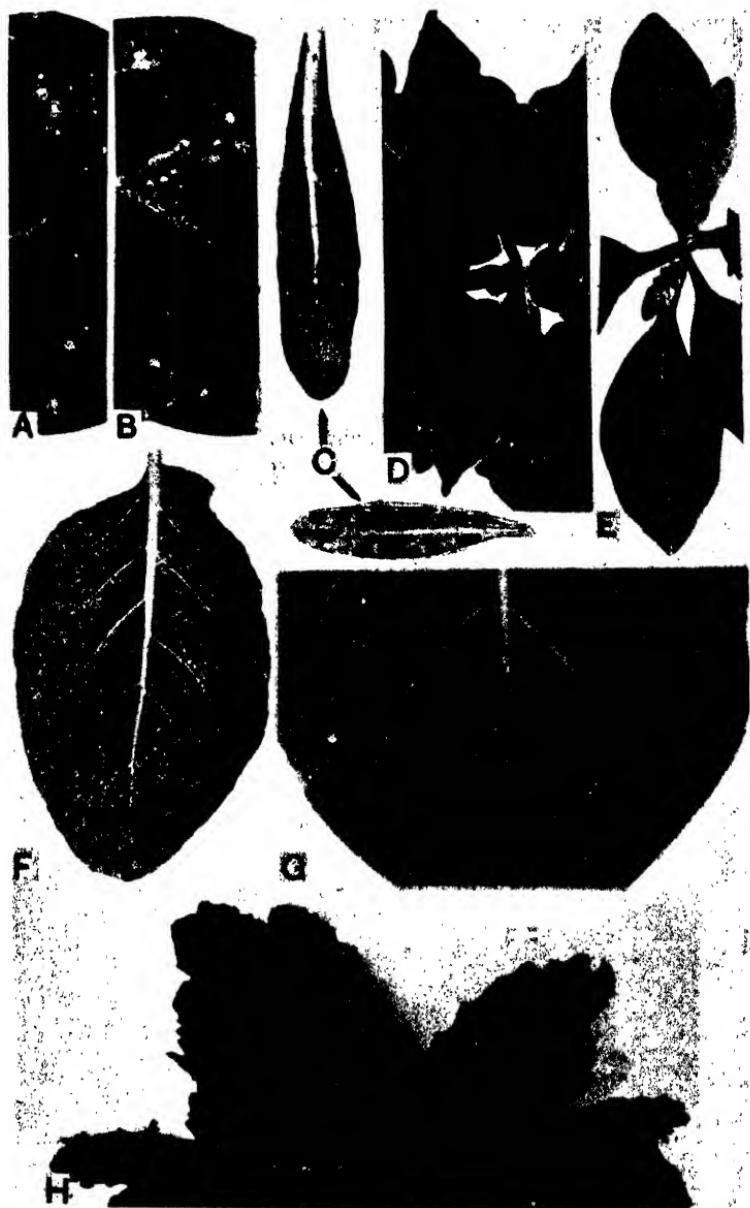
Nicotiana langsdorffii. In this plant the virus produces a pronounced, systemic mosaic. The mottling consists of pale yellow or light green patches on a darker background; the leaf edges are somewhat crinkled and there is no necrosis. Local lesions are not usually formed on this plant. There is some evidence that the virus may become attenuated after long periods of sub-culturing in this species.

Nicotiana tabacum, var. White Burley. In its reactions upon tobacco and *Nicotiana glutinosa*, this virus behaves in precisely the opposite manner to that in which tobacco mosaic virus (*Nicotiana Virus 1*) behaves on the same plant species. *Brassica Virus 1* produces necrotic local lesions on tobacco without systemic spread, while on *N. glutinosa* it does not produce local lesions, but gives rise to a marked mosaic mottling (see Figs. 1, B and 1, D).

Local lesions develop on the rubbed leaves of tobacco, five to six days after inoculation. These lesions are conspicuous and necrotic with a brown outer edge and a lighter centre. They enlarge considerably after their first appearance, frequently measuring 5 mm. in diameter. A similar type of local lesion is produced on tobacco by the feeding of infected individuals of the aphid *Myzus persicæ*. This is one of the few instances of the production of local lesions by the feeding of virus-infected aphides (see Fig. 1, A).

Nicotiana glutinosa. The incubation period of the virus in *N. glutinosa* is rather long, and it may be three weeks before signs of systemic infection appear. A mosaic mottle develops on the youngest leaves, and the disease finally takes the form of a bold mosaic with large, abnormally dark green patches interspersed with smaller patches of lighter green or yellow in the manner shown in Fig. 1, D. At the commencement of infection

8 DISEASES CAUSED BY BRASSICA VIRUS 1



there may be some necrosis, but this usually disappears as the disease progresses.

Diseases Caused by Brassica Virus 1

Cruciferæ

Brassica oleracea L. var. *capitata* L. Cabbage. The disease produced in cabbage by this virus has been called "cabbage ringspot." The symptoms consist of small black necrotic rings or spots, which may be sufficiently numerous to cover the leaf in the manner shown in Fig. 1, F. These lesions develop on the inoculated leaves of young cabbage plants, and in the glasshouse they develop sixteen to twenty days after inoculation. There are usually no symptoms on the youngest leaves of infected cabbage plants and there is no preliminary clearing of the veins as the first sign of infection. The first systemic symptom is the appearance of spots which become necrotic and black, taking on a ringspot appearance and frequently developing first on the undersurface of the leaves (see Fig. 1, G). Intracellular inclusions have not been observed.

Brassica oleracea L., var. *botrytis* L. Cauliflower and broccoli.

Diffuse systemic mottling develops when *Brassica Virus 1* is inoculated to cauliflower seedlings. This diffuse mottling in reality consists of small, pale green, roughly circular areas which stand out in marked contrast to the dark green background of the leaf. Vein-clearing and vein-banding symptoms have not been observed in cauliflower plants infected with this virus. Old infected plants of broccoli occur in the field showing the same type of necrotic ring which develops in affected cabbage plants.

FIG. 1. *Brassica Virus 1* (causing Cabbage ringspot disease).

- A. Local lesions on tobacco, White Burley, produced by the feeding of infected aphides (*M. persicae*).
- B. Local lesions on tobacco, White Burley, produced by rubbing with a petal of an infected wallflower.
- C. Two leaves of stock (*Matthiola*) systemically infected : note the raised green areas on the leaves.
- D. Systemic infection of *Nicotiana glutinosa*.
- E. Local lesions on *Solanum capsicastrum*, no systemic infection.
- F. Cauliflower, var. February, infected with the Californian virus. (After Tompkins.)
- G. Underside of young cabbage leaf showing the necrotic rings.
- H. *Arabis* sp. showing systemic infection.

Matthiola incana and *M. incana*, var. *annua*. Stocks. Stocks are frequently found naturally infected with *Brassica Virus 1* and the outstanding symptom is the production of variegated or "broken" flowers (see Fig. 2, A). Any variety of stock with self-coloured flowers produces, when infected, flowers with "broken" colour in the petals. Some of these white "breaks" in the mauve, pink or purple flowers are quite attractive; white flowers are unaffected. The symptoms on the leaves vary somewhat in virulence, but are usually quite characteristic. In a severely affected plant the edges of the leaves are twisted and the whole leaf is crinkled and curled; occasionally the leaves are almost tubular and may be twisted right round. The lower leaves show a yellow chlorosis with some necrosis of the midribs. The topmost or younger leaves exhibit a mottle of two shades of green with some yellow flecks. The darker green tissue frequently appears as small blisters raised above the lighter green (see Fig. I, C). As a rule there is less crinkling at the top of the plant. Occasionally leaves show a broad band of yellow down the centre with an edging of abnormally dark green. The first symptoms on young stocks, artificially inoculated, consist of a network of necrotic lines mainly following the veins on the young leaves. These leaves become distorted and wrinkled. Subsequent leaves are crinkled and mottled with some necrosis and with the apical edges rolled inwards. Symptoms develop twelve to fourteen days after inoculation under glasshouse conditions. The flower petals of infected stocks seem to contain the virus in high concentration and inoculation made to tobacco with the petals of a "broken" flower produce large numbers of the typical local lesions.

Cheiranthus Cheiri Linn. Common wallflower. Affected wallflowers are small and stunted, and the leaves usually, but not invariably, have a dark green and yellowish mottle. The most striking symptom, however, is the "breaking" of the colour in the flowers, chiefly in self-coloured varieties. The favourite blood-red variety seems particularly susceptible and the red flowers become striped and flecked with an unsightly yellow variegation. Inoculation to tobacco with one of these yellow-flecked petals produces the characteristic lesions shown in Fig. 1, B. Infected plants may show a curling and crinkling of the leaves very similar to that found in similarly diseased stocks.

Arabis sp. Rockcress. The symptoms show up clearly on the pale leaves of this plant and consist of a pronounced mottle of spots and ring-like markings of a pale yellow colour (see Fig. 1, H).

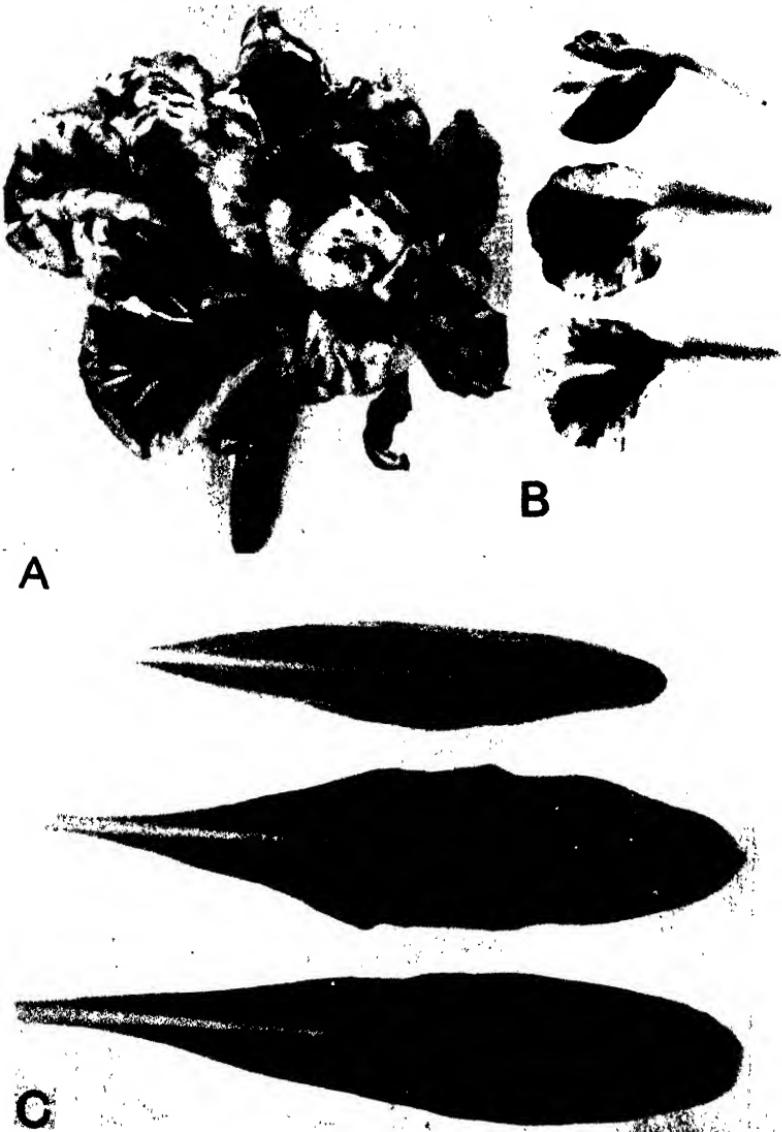


FIG. 2.

- Flower of stock (*Matthiola* sp.) infected with *Brassica Virus 1*: note the breaking of the flower colour.
- Flower petals of stock (*Matthiola incana*) var. Fiery Blood Red, infected with *Matthiola Virus 1* (Stock Mosaic): note type of break.
- Leaves of the same plant as that shown in B: note type of mottling induced.

(B and C, after Tompkins.)

Hesperis matronalis. Sweet Rocket. On this plant the disease produced by *Brassica Virus 1* is more severe than in the foregoing species, and death or severe crippling of the plant may ensue. The young leaves exhibit a marked mosaic mottle of dark green patches while the lower leaves are much crinkled and yellowed. The flowers are flecked or pencilled at the edges.

Geographical Distribution. *Brassica Virus 1* appears to be widely distributed. It occurs commonly in the British Isles, in Portugal, and probably in the United States of America.

BRASSICA VIRUS 2. Hoggan and Johnson

Synonyms. *Turnip Virus 1*, Hoggan and Johnson, 1935; possibly *Turnip Mosaic Virus*, Gardner, 1921.

The Virus

Thermal Death-point. The virus is inactivated by ten minutes' exposure to a temperature of 54° C.

Dilution End-point. The virus appears to lose infectivity at greater dilutions than 1 : 1,000.

Resistance to Ageing. The longevity of the virus in extracted sap at temperatures of about 20° to 22° C. is between twenty-four to forty-eight hours, and is always less than three days at that temperature.

Transmission. The virus is sap-inoculable and the insect vectors are the peach aphid, *Myzus persicae* Sulz., and the cabbage aphid, *Brevicoryne brassicae* (see pp. 526 & 538).

Differential Hosts

The diseases caused by this virus were first described by Hoggan and Johnson in 1935 (29), and although the virus seems similar in many ways to *Brassica Virus 1*, yet there are differences, and for the present it is considered better to treat it as a separate entity. The information, here given, is derived partly from the work of Hoggan and Johnson (29) and partly from some unpublished work by Smith and d'Oliveira.

N. glutinosa. Symptoms are similar to those produced by *Brassica Virus 1*, except that local lesions are formed on the inoculated leaves and there is more necrosis.

N. tabacum (var. White Burley). The local lesions formed by *Brassica Virus 2* on tobacco are smaller and more numerous than those formed by *Brassica Virus 1*. They are in the form of small

"water-soaked" spots rather than the large lesions produced by the latter virus.

N. langsdorffii. The virus apparently does not infect this species.

Diseases Caused by Brassica Virus 2

Cruciferæ

Brassica oleracea L., var. *capitata* L. Cabbage. There are no local lesions formed on the inoculated leaves and the first sign of infection is the development of a very faint mosaic mottling on the younger leaves. This mottle consists of large roundish blotches of slightly darker green against the normal lighter green background. These symptoms are best observed by holding the leaf up to the light. No necrosis has been observed.

Matthiola incana and *M. incana*, var. *annua*. Stocks. Infected stocks show a mottle of the leaves; this consists of dark green patches on the normal green colour of the leaf. The margins of the leaves are slightly waved. There does not appear to be any change in the flower colour.

Cheiranthus Cheiri Linn. Wallflower. The wallflower seems to be resistant to infection with *Brassica Virus 2*.

BRASSICA VIRUS 3. Tompkins

Synonym. *Cauliflower Mosaic Virus*, Tompkins, 1934.

The Virus

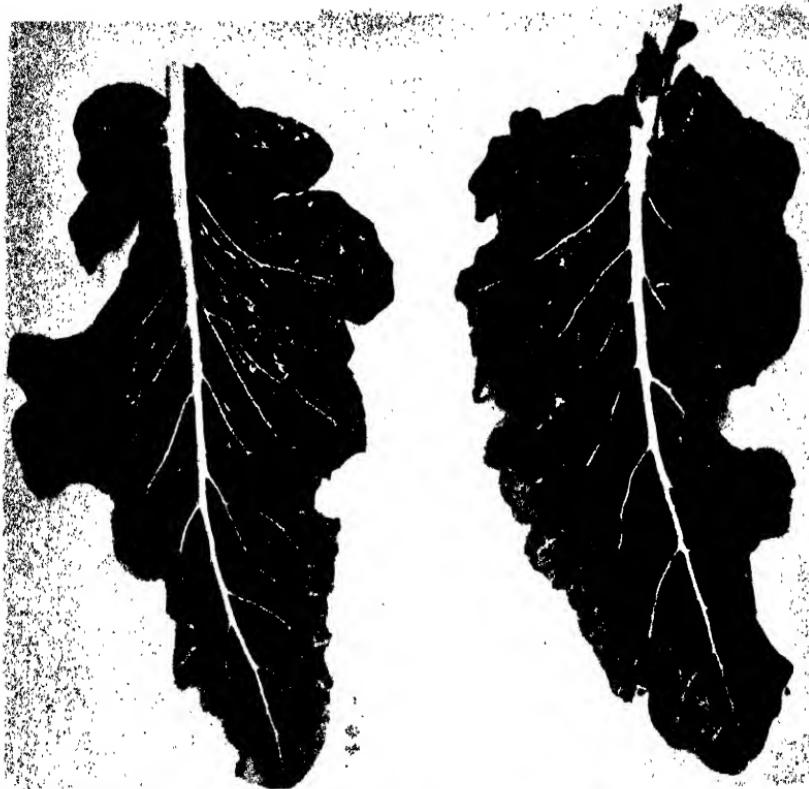
Thermal Death-point. Inactivation occurs at or near to 75° C. after exposure for ten minutes.

Dilution End-point. Tolerance to dilution is approximately 1 : 2,000.

Resistance to Ageing. In expressed juice stored at a constant temperature of 22° C. the virus remains infective for fourteen, but not for fifteen days.

Transmission. The virus is sap-inoculable, but with difficulty. It is more easily transmitted if a little carborundum powder is dusted on to the leaves or added to the inoculum. There is no evidence of seed-transmission. According to Severin (*in litt.*) the following species of aphid which breed on cauliflower under natural conditions in California are vectors of *Brassica Virus 3*; *Brevicoryne brassicae* L., the cabbage aphid; *Rhopalosiphum pseudobrassicae* Davis, the false cabbage aphid; *Myzus persicae* Sulz., the peach aphid (see pp. 526, 546, 538). The following species of

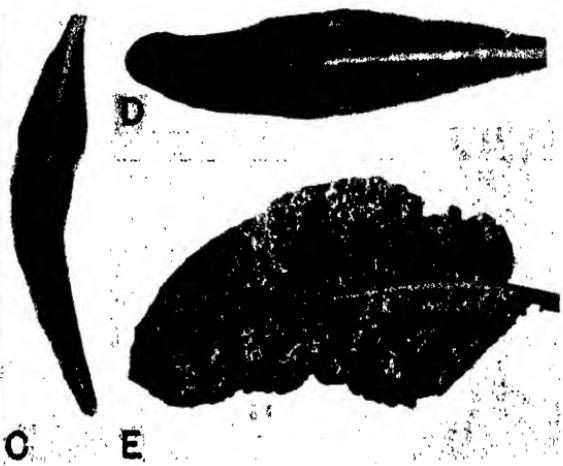
14 DISEASES CAUSED BY BRASSICA VIRUS 3



A



B



C

D

E

aphides which have not been found to breed on cauliflower under natural conditions, were shown by Severin experimentally to be capable of transmitting the virus :

- Aphis graveolens* Essig n. sp. Celery leaf aphid.
- Aphis apigraveolens* Theob. Celery aphid.
- Aphis middletonii* Thomas. Erigeron root aphid.
- Aphis gossypii* Glover. Cotton or melon aphid.
- Cavariella capreae* Fabr. Yellow willow aphid.
- Myzus circumflexus* Buckt. Lily aphid.
- Rhopalosiphum melliferum* Hottes. Honeysuckle aphid.

Differential Hosts

Brassica Virus 3 is not transmissible to *Nicotiana tabacum*, *N. glutinosa* or *N. langsdorffii*. It can be differentiated from *Brassica Virus 1* by the fact that it produces clearing of the veins of the youngest leaves as an initial symptom in cauliflowers followed by vein-banding and necrotic spotting (see Fig. 3, B).

Diseases Caused by Brassica Virus 3

Cruciferæ

Brassica oleracea L., var. *botrytis* L. Cauliflower. In cauliflower mosaic, infection is first manifested by pronounced vein-clearing which generally commences at or near the base of the leaf lamina on one or both sides of the midrib and gradually progresses upward till the entire veinal system is involved. At the onset of the disease one or several leaves may show vein-clearing, but frequently this symptom appears simultaneously on all leaves. Vein-clearing may persist for ten to twenty days, but gradually changes to vein-banding which consists of narrow, continuous, dark green areas parallel with and immediately adjoining the midrib and lateral veins. In the chlorotic areas between the veins are found numerous, irregularly-shaped, non-raised dark green

FIG. 3. *Brassica Virus 3* (causing Cauliflower mosaic).

- A. Mature leaves of cauliflower showing vein-banding.
- B. Seedling of February cauliflower, showing vein-clearing : *h*, healthy; *d*, diseased.
- C and D. Leaves of infected stock (*Matthiola incana*).
- E. Leaf of infected February cauliflower, showing necrotic type of symptom.

(B-E, after Tompkins.)

islands which produce the mottle effect. Later, small, irregular, necrotic lesions, often numerous, appear in the mottled area. These lesions, the colour of which remains unchanged by age, are different from the necrotic spots and rings characteristic of *Brassica Virus 1* (65). Symptoms on cauliflower are usually masked by temperatures above 65° F. (19° C.) (see Fig. 3).

Matthiola incana. Stock. *Brassica Virus 3* causes a rosette effect upon stock plants by the shortening of the internodes. Vein-clearing and slight upward curling of the leaf margins are characteristic of infection with this virus (see Figs. 3, C, 3, D). "Breaking" of the flower colour is not a symptom (compare *Brassica Virus 1* and *Matthiola Virus 1*).

Lunaria annua. Honesty. Affected plants show vein-clearing, followed by a systemic mottle. In advanced stages of the disease the plants are stunted with ruffled and distorted leaves.

Host Range. Tompkins (65) gives the following list of cruciferous plants susceptible, either naturally or experimentally, to infection with *Brassica Virus 3*: kale (*Brassica oleracea* L., var. *acephala* DC, brussels sprouts (*B. oleracea* L., var. *gemmaifera* Zenk.), red and white varieties of cabbage (*B. oleracea*, var. *capitata* L.), asparagus or sprouting broccoli (*B. oleracea* L., var. *italica* Plenck), wild mustard (*B. campestris* L.), charlock (*B. arvensis* L.), shepherd's purse (*Capsella bursa-pastoris* Moench), jointed charlock (*Raphanus raphanistrum*). The experimental host range, as determined by inoculations, includes fifty-one vegetable varieties, three ornamental plants and five wild cruciferous plants.

Geographical Distribution. *Brassica Virus 3* has so far only been recorded from the United States of America by Tompkins (65) and from England by the writer.

Control. No indication of resistance has been found in any of the cauliflower varieties of commercial importance in California. Until resistance is ultimately obtained through a combination of selection and breeding processes, Tompkins (65) suggests that considerable damage to the crop can be avoided (1) by eradication of wild cruciferous and other weed plants which occur in or near cultivated fields on which the various *aphis* vectors breed naturally, and (2) by systematic dusting or spraying of the plants at regular intervals to keep down *aphis* infestation. In California treatment should commence while the plants are in the open field seed-beds, especially in those coastal areas where cauliflowers are grown throughout the year, and be continued after transplanting until the plants are well along towards maturity.

BRASSICA VIRUS 4. Clayton

Synonyms. *Crucifer Mosaic Virus*, Clayton, 1930. Probably the same as turnip and rutabaga mosaic virus. This virus may prove later to be the same as *Brassica Virus 2*.

The Virus and its Transmission. No studies appear to have been carried out on the physical properties of this virus. The virus is sap-inoculable and there is no evidence of transmission by the seed. According to Clayton (18) the insect vector is the cabbage aphis, *Brevicoryne brassicae*, and not the peach aphis, *Myzus persicae*.

Diseases Caused by Brassica Virus 4**Cruciferæ**

Brassica chinensis L. Chinese cabbage. Plants of this variety are highly susceptible to the virus; affected plants show a distinct mottling of the leaves with light and dark green areas. The light green areas usually adjoin the veins from which they may extend so as to include a considerable area of the leaf surface between the veins. Another common symptom is the ruffling and distortion of the leaf surface with dark green patches on the raised areas. The leaf margins are frequently irregular, giving the plant an unsymmetrical appearance. In addition the entire plant may be dwarfed and the flower stalks and numbers of blossoms may be considerably reduced. At high temperatures diseased plants are much stunted and show streak symptoms.

Brassica alba Rabenh. Cultivated white mustard. *B. nigra* Koch, Wild black mustard. Leaves of affected plants show faint mottling, but more severe symptoms develop at high temperatures when the plants are yellow and much stunted and usually develop streak symptoms. Mustard is very susceptible to infection with this virus.

Brassica rapa L. White turnip. This plant is readily infected and develops a pronounced leaf mottling. At high temperatures diseased plants are yellow and stunted, but do not develop streak symptoms. Although readily infected this species is less severely injured by the virus than the two foregoing plants.

Brassica napobrassica Mill. Swede. The susceptibility and symptoms of this species are very similar to those of the turnip.

Brassica napus L. Rape. Symptoms consist of mottling and some stunting, this plant is less easily infected than the turnip or swede.

Brassica oleracea L., var. *gemmaifera* Zenker. Brussels sprouts. Some affected plants of this variety show the usual mosaic mottle, but others develop round yellow blotches scattered evenly over the leaves and not raised. Later the tissues round the edges of the yellow areas die out, giving a ringspot effect. Infected plants at high temperatures are stunted by the disease, but at lower temperatures it is not uncommon for new growth to be free of mottling. On the whole brussels sprouts are rather resistant to infection with this virus.

Brassica oleracea L., var. *botrytis* L. Cauliflower. The symptoms are rather similar to those on brussels sprouts, consisting of yellow spots scattered over the green leaf. There is not, however, the same tendency for the edges round the spots to die out.

Brassica oleracea L., var. *capitata* L. Cabbage. According to Clayton (18) the cabbage is resistant to infection with *Brassica Virus 4*.

Geographical Distribution. The virus has been recorded from the United States of America, and what is probably the same entity has been studied by Kaufmann (32) in Germany.

MATTHIOLA VIRUS 1. Tompkins

Synonym. *Stock Mosaic Virus*, Tompkins.

The Virus and its Transmission. There is no information at present on the properties of *Matthiola Virus 1*.

The virus is sap-transmissible and infection is more easily obtained by the addition of carborundum powder to the inoculum. There is no evidence of seed transmission. The insect vectors are the aphides, *Myzus persicae* Sulz., *Brevicoryne brassicae* and *Rhopalosiphum pseudobrassicae*, the turnip or false cabbage aphis. The last-named insect breeds upon stocks under natural conditions in America and is probably the chief vector (see p. 546).

Differential Hosts.

Matthiola Virus 1 is not transmissible to cabbage, cauliflower or *Nicotiana* spp., and the only known host at present is the stock, *Matthiola incana*.

Disease Caused by Matthiola Virus 1

Cruciferae

Matthiola incana. Stock. *Stock Mosaic*. The virus causes a definite mottling on stock leaves, the dark green islands being

quite pronounced and conspicuous in contrast with the light green areas. No necrotic spotting or other leaf symptoms have been observed. Under field conditions the plants are stunted in varying degree, with slight shortening of the internodes. Where plants are grown for seed, the pods are greatly reduced in length and size. In addition to leaf mottling, which may not always be visible on the foliage of infected plants in the field, "breaking" of the flower colour is perhaps the chief and most reliable symptom. Complete or sectorial "breaking" may occur; often on the same plant, but on different racemes, the flowers may be normal in colour or may be "broken." The petals of "broken" flowers are undersized (see Figs. 2, B and 2, C).

In California all varieties of stocks appear to be susceptible to infection. Entire fields of stocks intended for cut flowers are frequently a total loss as "broken" flowers are not wanted by the trade.

Cheiranthus Cheiri Linn. Wallflower. A virus isolated from wallflowers in Cambridge, England, seems to be very similar to, if not identical with, *Matthiola Virus 1*. In the field the diseased wallflowers presented a discrete mottling of the leaves, and this condition was reproduced in young plants of wallflower by sap-inoculation in the glasshouse; these inoculated plants bore "broken" flowers. A mottling was also produced on the leaves of *Matthiola incana*, and these plants bore flowers with "broken" colours. Cabbage, *Nicotiana glutinosa* and *N. langsdorffii* showed no reaction when inoculated with the virus. As regards tobacco, var. White Burley, there seems to be some evidence that the inoculated leaves may show small necrotic lesions from which the virus can be recovered (d'Oliveira, unpublished work).

Geographical Distribution. This virus appears to be widespread in California, and a disease of stocks, probably caused by the same virus, has been observed by the writer in England.

Other Undifferentiated Viruses Affecting Cruciferous Plants

Cochlearia Armoracia Linn. Horseradish. A virus disease affecting horseradish has been described in Washington County, Oregon, U.S.A. (16). The foliage of infected plants is stunted and the leaf blades are strikingly segmented in a fern-like manner. The young leaves exhibit a prominent mosaic mottle, characterised by interveinal pale green areas interspersed with dark green. Clearing of the veins is not noticeable. Old infected leaves tend

to develop black, elongated lesions in the epidermis and outer cortex of the petioles. At digging time most of the affected plants are dwarfed and yellowed. The roots are small and unsightly because of the rough scaly surfaces, pitted texture and frequent dark streaks. The virus has been transmitted by mechanical inoculation from horseradish to turnip and mustard.

Lunaria annua. Honesty. A mosaic disease of this plant has been recorded in England by Ogilvie (44) and by Tompkins (*in litt.*) in California. Tompkins states that for several years in succession this virus has wiped out entire plantings of *L. annua* in a small coastal valley at Montara, south of San Francisco. The writer has also received mosaic specimens of this plant from Wales. The virus is different from *Brassica Virus 1*, since it is not transmissible to cabbage or cauliflower. It causes systemic infection on *N. glutinosa*, and on Turkish tobacco small local necrotic lesions are formed. Its affinities, if any, with the other mosaic viruses affecting cruciferous plants are not known. Diseased *Lunaria* plants show a coarse mottling of light green on a darker background.

Raphanus sativus. Radish. Mosaic. A virus causing a mosaic mottling on wild and cultivated radish has been observed by Tompkins at San Pablo, near Berkeley, and at Colma, California. The mottling takes the form of patches of dark green on a lighter background mainly in the neighbourhood of the veins; this occasionally gives a vein-banding effect.

Raphanus sp. Chinese radish. Mosaic. A mosaic virus affecting Chinese radish has also been observed by Tompkins at Salinas, California. He considers this to be a different virus from that described above on radish. It produces a coarse type of mottling with raised dark green islands on the leaves together with some distortion.

Brassica Napus L. Rape. A virus disease of rape and swedes has recently been described from Kiel in Germany. In one variety of rape the symptoms consist of a twisting and crinkling of the young leaves and premature dying of the old leaves and of many plants before or during the winter. In diseased plants which survive the winter, retardation or inhibition of growth occurs in spring and summer. Swedes and another variety of rape are attacked to a less intensive and extensive degree. There is no twisting of the leaves, but mosaic marking and a crinkling of the leaf surfaces with fissures at the edges are particularly noticeable. The plants are often crippled but only

rarely killed. Artificial inoculation is easily accomplished with both forms of rape and with swedes, but it fails with other crucifers. *Lygus pratensis* L. (see p. 468), collected on diseased rape, readily transmits the virus to healthy rape and swedes.

Most of the rape plants acquire the infection in summer and autumn, while swedes become infected in the spring and maintain the virus till autumn. Even in the absence of swedes, *L. pratensis* can retain the virus during the interval between the death of the old plants and the sprouting of the new ones (82).

BETA VIRUS 1. Boncquet and Hartung

Synonyms. *Sugar Beet Virus 1*, J. Johnson's classification; *Sugar Beet Curly-top Virus*, Boncquet and Hartung, 1915; *Sugar Beet Curly-leaf (Virus)*, Ball, 1909; *Western Yellow Blight Virus*, *Tomato Yellows Virus*.

The Virus

Although this virus is not easily sap-inoculable, methods of study evolved by Bennett (5) and Severin and Freitag (60) have enabled certain of its properties to be determined.

Resistance to Various Chemicals. Alcohol and Acetone. Two-hour treatments with 90 per cent. alcohol and with lower concentrations have no appreciable effect on the virus. Absolute alcohol reduces but does not destroy infectivity in a two-hour period. A two-hour treatment with acetone has no apparent effect at any concentration. The virus appears to be still active after fifty-six days in 50 and 75 per cent. alcohol and in 75 per cent. acetone although activity is considerably decreased.

Miscellaneous Reagents. The virus exhibits considerable resistance to the lethal action of a number of common disinfectants, it is not inactivated by copper sulphate (1 : 200), bichloride of mercury (1 : 50), formaldehyde (1 : 100), and carbolic acid (1 : 25).

The expressed juice from beet and a number of other species of plants is able to cause inactivation of the virus in periods ranging from thirty minutes to more than fourteen days, depending on the species of plant from which the juice is extracted.

Thermal Death-point. The thermal inactivation point of the virus lies between 75° and 80° C. A ten-minute exposure to a temperature of 76° to 79° C. inactivates the virus.

Effect of pH. No virus can be recovered from liquids having a pH value of 2.9 or lower. Alkaline reaction of the medium as high as pH 9.1 does not inactivate the virus in two hours.

Dilution End-point. Bennett (5) recovered virus from dilutions of 1 : 1,000 in experiments in which one artificially-fed leafhopper was placed on each plant and from dilutions of 1 : 20,000 in experiments in which ten leafhoppers were placed on each plant. Severin and Freitag (60) found that the virus from crushed infective leafhoppers would stand a dilution of 1 : 24,000, but the virus from centrifuged beet extract was inactivated at dilutions higher than 1 : 1,000.

Resistance to Ageing. The resistance of the virus to ageing in a liquid medium depends considerably on the medium in which the virus is preserved. In filtered and unfiltered beet leaf juice the virus can be recovered after seven days. It can be recovered from unfiltered water washings of alcohol precipitate of leaf juice after fourteen days and from filtered water washings of the same after twenty-eight days.

Desiccation. The resistance of the virus to desiccation also depends somewhat on the medium in which it is kept. Virus can remain active ten months in dried phloem exudate, five months in alcoholic precipitate of phloem exudate, four months in dried beet tissue, two months in alcoholic precipitate of beet leaf juice and beet root juice, and six months in dried beet leafhoppers.

Filterability. The virus passes the ordinary filters such as Berkefeld V, N, and W, the Mandler medium and fine grades, and the Chamberland filter condensates L₁, L₃, L₅, L₇, L₉ and L₁₃.

Attenuation and Restoration of Virulence. It was shown originally by Carsner and Stahl (11) that passage of the virus through *Chenopodium murale* L. reduces its virulence. *Rumex crispus* L., *Suaeda moquinii* Greene, and certain resistant types of sugar beet also have an attenuating effect. Such attenuated strains of the virus can be restored to their original virulence by passing them through chickweed, *Stellaria media* L. (37).

Bennett has shown (5) that a somewhat similar type of attenuation can also be induced by heating the virus for ten minutes at 76° to 79° C.

Transmission. Although it is very difficult to transmit the virus by sap-inoculation, yet this can be done, using specialised methods. Severin (57) induced infection in beets by making repeated punctures with insect pins into the crown through drops of expressed beet juice. Bennett (4) succeeded in infecting fourteen out of 124 plants by using as his source of inoculum the phloem exudate from the cut surfaces of diseased beets. It appears that the virus is confined to the phloem tissues of infected beets, and

that the other tissues are actually toxic to it. The specific insect vector, *Eutettix tenellus* Baker (see p. 480) is primarily a phloëm feeder, and this is possibly one important reason for its ability to transmit the virus. As the mouth-parts are inserted in the tissue the insect lays down a sheath of apparently gelatinous material which completely encases the stylets. Bennett suggests that this sheath may seal off all cells penetrated that are external to the phloëm and thereby protect the virus as it is passed into or drawn out of the phloëm by the leafhopper. There appear to be two types of transmission of the virus by the insect; in the first, infection is brought about after short periods of feeding on diseased and healthy beets, this is considered to be purely mechanical transmission by contaminated mouth-parts. The other type of transmission necessitates a delay in the development of infective power within the insect. This period may vary from twenty-one to twenty-four hours.

Differential Hosts

Beta Virus 1 has a very wide host range and the symptoms it produces on most of these plants have a general similarity. It is therefore not easy to pick out any particular species as indicator plants, but since *Datura Stramonium* and *Nicotiana tabacum* are easily grown and are useful as differential hosts for many other viruses, these two have been selected for this purpose.

Nicotiana tabacum. Tobacco, var. White Burley. The first symptom to develop in an experimental infection is a clearing of the veins. A marked stunting of the infected plants follows together with a shortening of the internodes. The youngest leaves are dwarfed and outwardly cupped.

Datura Stramonium. The youngest leaves of infected plants are dwarfed and the venation of the older leaves may be transparent (58).

Diseases Caused by Beta Virus 1

Much of the information on the different diseases caused by this virus is derived from the work of Severin and his colleagues (58).

Cruciferae

Matthiola incana. *M. incana*, var. *annua*. Stocks. Diseased stocks are stunted with shortened internodes and numerous axillary shoots bearing linear leaves at the apices of the branches. The leaves of the axillary shoots are sometimes twisted and often

the margins of the leaves are rolled or curled inwards. The veins on the lower surface of the leaves are distorted with protuberances resembling tiny warts. The apices of the leaves of some plants become purple or yellow. The lower leaves of plants in an advanced stage of the disease are dry, and the leaves near the terminal end of the shoots become white. Brown droplets of liquid exude from the blades, petioles and stem. The flower stalks from numerous axillary shoots form a dense cluster and the flowers are often malformed with dried petals.

Cochlearia Armoracia. Horseradish. Infected plants show an inward curl of the leaves with exudation of sap from the petioles of some of the leaves (see Sugar Beet, p. 25). Later the foliage may turn yellow. The roots of horseradish plants infected early in the season are dwarfed and brittle. A cross section of a diseased root shows darkened rings and bundles in the interior, while a longitudinal section shows the dark discolorations extending lengthwise through the root.

Raphanus sativus. Radish. Infected radishes show an inward curl of the leaves with outstanding veins, plants may be stunted and show yellowing of the leaves.

Brassica spp. The Cabbage, Cauliflower and Turnip are all susceptible to infection. The plants, however, do not show any very characteristic symptoms of infection except for occasional exudation of sap and slight inward rolling of the leaves.

Barbarea vulgaris. Cress, vars. Fine Curled and True Water. The youngest leaves of the first-named variety become curled with faint indications of transparent venation. Affected plants of the second variety show a shortening of the petioles at the terminal end of the shoots, an inward roll of the leaflets, and sometimes elevations on the lower surface of the leaves.

Capsella bursa-pastoris. Shepherd's Purse. This weed is sometimes found naturally infected. Affected plants are stunted with twisted seed stalks usually bearing malformed seeds near the terminal ends.

Violaceæ

Viola tricolor, var. *hortensis*. Giant Trimardeau Pansy. *Viola cornuta*, var. "Apricot Queen." The most conspicuous symptom of the disease in the above plants is the dense cluster of chlorotic secondary shoots arising from the axil of the leaves near the tips of the branches of the stunted plants. The margin of the leaves is rolled inwards or cupped along the midrib, or the tips of

the leaves are rolled towards the petioles. The youngest leaves show cleared or transparent veinlets. The veins are wavy and bear small protuberances or papillæ. Dwarfed linear leaves surround the flower buds near the tip of the secondary shoots. The flower buds are often sessile, sometimes with a short peduncle. The flowers are dwarfed and frequently dry.

Polygonaceæ

Fagopyrum esculentum. Common buckwheat. The first symptoms to appear on this species are blister-like elevations on the youngest leaves, then the margins of these leaves roll inwards with the tissue sunken between the lateral veins and the twisted petioles. In the later stages of the disease the leaves are dwarfed and often cupped outward. Death of the plant frequently ensues.

Rheum rhabonticum. Rhubarb, var. Giant Crimson Winter. This plant is susceptible to infection, but shows no very characteristic symptoms.

Rumex scutatus. French large-leaved sorrel. Plants of this species when experimentally infected develop, in the later stages of the disease, wart-like protuberances on the lower surface of the leaves.

Weed Hosts. The following weeds belonging to the Polygonaceæ have been found naturally infected with *Beta Virus 1* (curly-top virus); *Polygonum aviculare*, wire grass; *P. muhlenbergii*, swamp smartweed; *P. amphibian hart-wrightii*, water smartweed; *P. lapothifolium*, common knotweed; *P. persicaria*, lady's thumb.

Rumex crispus, the curly dock, when infected with the virus develops wart-like protuberances on the under surface of the leaves in a similar manner to *R. scutatus*. These protuberances are a common symptom of the disease in most plants of the Chenopodiaceæ, but in plants of other orders they have only been observed in the two species mentioned above and in tomato.

Chenopodiaceæ

Beta vulgaris. Sugar beet. *Curly-top*. (Fig. 4, A). Severin (59) classifies the main symptoms of curly-top on sugar beet under the following heads, leaf-curling, blister-like elevations on the leaves, transparent venation, protuberances on the lower surface of leaves, exudations from petioles, midribs or veins and yellowing. Of these symptoms the most reliable for diagnostic purposes are the clearing or transparency of the minute veins on the youngest



FIG. 4. *Beta Virus 1* (Sugar beet curly-top virus).

A. Infected sugar beet.

B. Infected bean (*Phaseolus vulgaris*). (After Severin.)

or innermost leaves in the early stages of the disease and wart-like protuberances on the lower surface of the leaves in the later stages of the disease.

The earliest symptom of curly-top to appear is an inward rolling of the lower and outer margin of the youngest leaves. Later the entire blade may show a pronounced inward curling towards the midrib. Small blister-like elevations may develop on the leaves of beet seedlings, shortly after infection, but this is not an invariable symptom. A more constant symptom is the transparent network of minute veins generally occurring on the innermost or youngest leaves of the beet. At the beginning, this symptom may be confined to a portion of the youngest leaf, but in a few days the entire leaf is affected. The other reliable symptom is the roughened appearance of the lower surface of the leaves, developing usually after the veinlets have become transparent. A closer examination of this roughened condition upon its first appearance reveals numerous small elevations on the veins resembling tiny warts. As the disease progresses, nipple-like papillæ and knot-like swellings resembling galls develop here and there on the distorted veins. The diseased leaves are dark, dull green in colour, thick, crisp, and brittle. Occasionally an infected plant may show a few drops of clear viscid liquid exuding from the petioles, midrib or veins on the lower surface of the leaves. Later this liquid becomes black and sticky and upon drying forms a brown crust. An increase in the number of rootlets is also a symptom of a badly-diseased beet; this condition is sometimes described as "hairy" or "woolly" root. A cross-section of an affected beet often shows black concentric rings which alternate with light areas. A longitudinal section shows the dark discolouration extending lengthwise through the beet.

Similar symptoms develop on mangel wurzel, red or garden beet and Swiss chard (*Beta vulgaris cicla*) when infected with *Beta Virus 1*.

Histopathology. This virus induces phloëm degeneration in infected beets. One or more of the primary sieve tubes differentiate before the characteristic degenerative changes become perceptible in the phloëm. Pericycle or phloëm-parenchyma cells adjacent to the sieve tubes undergo hypertrophy and die. These phenomena have been termed primary hypertrophy and primary necrosis. Cells further removed from the sieve tubes are stimulated to growth and division, a phenomenon designated as *primary hyperplasia*. A large proportion of the hyperplastic cells undergo a series of

changes characteristic of differentiating sieve tubes : they develop slime bodies and plastids ; their nuclei and slime bodies disintegrate ; the cytoplasm is reduced in amount and the walls thicken. Sieve plates seem not to complete their development, for callus fails to develop. Companion cells may or may not be associated with these sieve tube-like cells. This tissue, in which sieve tubes predominate, is so striking in appearance as to identify the disease readily in its early stages.

The abnormal sieve tubes and their companion cells later die and collapse, a process termed secondary necrosis. The near-by parenchyma cells undergo secondary hypertrophy and hyperplasia, resulting in proliferations resembling callus.

These facts strongly suggest that the virus moves in the phloem through the mature sieve tubes (22).

Spinacia oleracea. Spinach. The youngest leaves of affected spinach plants show a clearing or transparency of the small veins, but this symptom is often difficult to distinguish from the normal venation. The leaves may develop an inward curl or roll, as in the prickly seeded spinach, or an outward roll or curl towards the petiole, as in Virginia savoy spinach. Later the young plants turn yellow and die.

Weeds and Shrubs of the Chenopodiaceæ. A number of different weeds and shrubs are susceptible to infection with the virus, and the following have been found naturally infected in the field : *Atriplex bracteosa*, bractscale ; *A. rosea*, red scale or orache ; *A. argentea expansa*, silver scale or fog weed ; *A. patula hastata*, spear scale or spear orache ; *Chenopodium leptophyllum* ; *C. murale*, nettle-leaf goosefoot ; *C. ambrosioides*, Mexican tea ; *Salsola kali tenuifolia*, Russian thistle.

Geraniaceæ

Pelargonium hortorum. Geranium. The leaves of young shoots on affected plants are cupped inwards with sinuous veins, cleared veinlets, and protuberances on the lower surface. The older leaves are chlorotic between the veins, while the area in the vicinity of the veins remains green. The apical leaves on old branches show protuberances on the lower surface, with dwarfed chlorotic leaves developing from the nodes. The older branches are yellow instead of green.

Tropæolaceæ

Tropæolum majus. Nasturtium. The outer or older leaves of naturally infected plants are usually yellow. Numerous

secondary shoots are present with dwarfed, cupped and sometimes puckered leaves with distorted veins. The dwarfed leaves are sometimes dry; occasionally the petiole below the blade is withered, while the remainder of the petiole is green. The leaves near the apices of the secondary shoots are dwarfed; they have small blades with the margins rolled inwards. The sepals and petals of immature flowers are withered or dry. The flower buds usually are dwarfed and chlorotic and fail to expand. The pistil is sometimes enlarged.

Cucurbitaceæ

Cucurbita pepo, *C. maxima*, *C. moschata*. Pumpkin, squash, vegetable marrow. The first symptoms to appear are puckering and outward cupping of the newly developing dwarfed leaves. In some varieties the cupping continues until the leaves are almost globular in shape. Transparent venation is often discernible, sometimes accompanied by mottling of the somewhat older leaves. The dwarfed, cupped leaves and petioles are often dark green, with the stems darker green, compared with healthy plants of the same age. The flowers of infected plants are also dwarfed and tend to drop from the plants. The calyx in the larger flowers is present, but sometimes no corolla develops. Occasionally the youngest dwarfed leaves may show a slight cupping or may be normal in shape. Successive stages of discoloration, from mottling of the older leaves to a decided yellowing of the youngest leaves, occur in some varieties. The yellow discoloration gradually develops between the lateral veins in the older leaves, while the area in the vicinity of the midribs and lateral veins may retain the green colour for a time.

Citrullus vulgaris. Watermelon. The first leaves of the terminal shoots of infected watermelons show a slight puckering and outward curling. The youngest leaves are deep green, in contrast with the yellow of the older leaves. Naturally infected watermelons are stunted and yellow, with dwarfed leaves at the terminal end of the runners.

Cucumis sativus. Cucumber. Cucumbers, infected with the virus after the first true leaves develop, are stunted. The youngest leaves become deep green in colour while the older leaves turn yellow. The yellowing begins at the margin of the leaf and progresses between the lateral veins toward the midrib with a green area along the veins, and at the base of the leaf. Later, the stem often bends near the surface of the soil and the plant wilts

and dies. The leaves at the terminal ends of the runners of naturally infected cucumbers are dwarfed, sometimes slightly cupped and densely clustered together. The fruit is dwarfed and often malformed.

Cucumis melo reticulatus. Muskmelon. Different varieties of muskmelons, both experimentally and naturally infected, show no very characteristic symptoms. The dwarfed youngest leaves of the stunted plants are sometimes puckered with margins slightly turned down. In the later stages of the disease the leaves become yellow. The flowers are also dwarfed and often become dry before the petals expand. In extreme cases of dwarfing, the flowers are reduced to small round knobs.

Leguminosæ

Phaseolus vulgaris. French bean, string bean. The first symptoms to appear, one to two weeks after infection, are a puckering and outward cupping of the newly developing leaves, with a clearing or transparency of the minute veins. The youngest leaves are decidedly dwarfed and of a darker green. In naturally infected beans of the Small White variety the cupping of the three leaflets sometimes continues until each leaf resembles a small green ball (see Fig. 4, B). An early-infected bean of this variety usually bears no pods, though occasionally a few small twisted pods may be present.

Vicia faba. Broad bean, horse bean. Three varieties of broad bean experimentally infected with curly-top showed an inward curl, blister-like elevations and transparent venation of the youngest leaves.

Vigna sinensis. Cowpea. There appear to be no very characteristic symptoms in affected cowpeas, though the plants are stunted and the leaves slightly yellow.

Vicia sativa. Spring vetch or tare. *V. aliopurpurea*. Purple vetch. *V. villosa*. Winter vetch. In diseased vetch plants the youngest leaflets nearest the petioles of the compound leaves are often rolled inward along the midrib while the terminal leaflets are malformed. The petiole may be bent downwards or the petiole and midrib may show a spiral twist.

Medicago hispida. Bur clover. This plant, which is valued as dry fodder on the plains and foothills of California, is susceptible to infection.

In the diseased plant the three leaflets fold along the sinuous

distortions of the midrib, and transparent venation is evident on the youngest leaves.

Melilotus alba. White sweet clover. The youngest leaflets of affected plants are cupped outward along the midrib with faint indications of transparent venation. Similar symptoms are produced in *Melilotus indica*, bitter clover; *Trifolium repens*, white Dutch clover; *T. hybridum*, Alsike or Swedish clover; *T. incarnatum*, crimson clover; and *T. pratense*, red clover.

Umbelliferæ

Petroselinum hortense. Parsley. Although susceptible to infection with the virus, affected parsley plants exhibit no visible foliage symptom of the disease.

Fæniculum dulce. Florence Fennel. Plants experimentally infected show a shortening of the petioles of the youngest leaves, with curled thread-like leaflets. The petioles of slightly older leaves often droop.

Compositæ

Zinnia elegans. Common zinnia. The internodes near the apices of the branches are shortened, with chlorotic secondary shoots arising from the axils of the leaves. The flowers are dwarfed with the petals reduced in number. Experimentally infected plants grown in the glasshouse may show transparent or cleared veinlets on the youngest leaves. This symptom has not been observed in the field.

Cosmos bipinnatus. Common cosmos. The internodes of infected plants are shortened towards the tips of the stems, and the apices of the branches and secondary shoots become yellow. Leaflets are often curled and twisted and the petioles are bent downwards; sometimes two adjacent petioles are bent parallel to the stem. The flower buds on the secondary shoots are dwarfed and surrounded by a cluster of small chlorotic leaves.

Coreopsis tinctoria. Calliopsis. Infected calliopsis plants are stunted compared with the normal but show no foliage symptoms.

Helichrysum bracteatum. Strawflower. Numerous secondary shoots develop towards the tips of the branches. The older leaves of the secondary shoots are curled outwards with small protuberances on the distorted veins, while the younger leaves are linear and sometimes twisted into a spiral. The flowers near the tips of the branches are dwarfed. The older leaves of the secondary shoots may show cleared or transparent veinlets.

Solanaceæ

Lycopersicum esculentum. Tomato. The disease caused in tomato plants by *Beta Virus 1* (curly-top virus) is usually known as *tomato yellows* and previously as *western yellow blight* of tomato.

In the field, the principal symptoms of the disease are an inward rolling of the leaflets along the midrib; the petiole and midrib frequently curve downwards, giving the leaf a drooping but not wilting appearance. The leaves become somewhat thickened and crisp. Later they assume a yellow colour with purple veins. This purpling of the veins, however, cannot be considered a reliable symptom. The stems become hollow owing to the drying of the pith. With the first appearance of these foliage symptoms the plant stops growing and assumes an erect or rigid habit. These foliage symptoms are probably due to the abnormal accumulation of carbohydrates in the affected plants, which in turn results from the stoppage of vegetative growth. If small fruits have been formed, they ripen prematurely and the seeds are abortive. A decay of the roots occurs, usually beginning at the tips of the smaller roots. The plant finally dies, the stems and leaves turning brown.

Under glasshouse conditions the first reliable symptom to appear is transparent venation. An inward curl of some of the leaflets occurs, especially in older plants. The purple venation is often absent under these conditions. White excrescences sometimes appear on the veins resembling somewhat the wart-like protuberances on curly-top beets. A yellowing often develops between the veins, while these remain green. A marked stunting occurs in plants affected while still young. Later the entire plant turns yellow and dies.

Solanum tuberosum. Potato. Potato plants naturally infected are stunted with yellowish, inward-rolled leaflets and sometimes a bending of the petioles. Potatoes in an advanced stage of the disease frequently develop dwarfed shoots in the axils of the leaves near the tip of the plant. Affected potato plants finally turn yellow and die. The following varieties have been experimentally infected by Severin: American Wonder, British Queen, Idaho Gem, Idaho Rural, Improved Early Rose, White Rose and Wisconsin Pride.

Capsicum frutescens. Pepper. As a general rule, an inward curl of the youngest leaves and an outward cupping of the somewhat older leaves are characteristic symptoms. In experimentally infected plants the veinlets of the youngest leaves become

transparent, but this has not been observed in naturally infected plants. Minute swellings develop on the network of cleared veinlets resembling somewhat the warty protuberances on the leaves of sugar beets in an advanced stage of the disease. Severin records the following varieties of peppers as naturally infected : Anaheim Chili, Paprika, Pimiento and Mexican Chili.

Petunia hybrida. Petunia. Diseased petunia plants are stunted with dwarfed cupped leaves and numerous secondary shoots. Protuberances on the veins on the lower surface of the leaves of secondary shoots and apical ends of the branches give the veins a roughened appearance. The corolla of the flowers often fails to expand and becomes dry. In the later stages of the disease the plant becomes yellow and dies.

Geographical Distribution. The curly-top disease of sugar beet was first recognised as of major importance in 1899 in California. Since that time frequent and often very destructive outbreaks have occurred in practically all the sugar beet areas west of the Rocky Mountains except a few districts in the fog belt along the Pacific coast. Its occurrence in serious amounts east of the Continental Divide has apparently been restricted to certain sections in the south-west, such as New Mexico, Western Texas, and Southern Colorado. The beet areas in Northern Colorado, east of the Rocky Mountains, as well as those in Nebraska, Kansas, Wyoming and South Dakota, have been free of the disease or have shown only sporadic incidence (9). In 1934 there was an unprecedented spread of the disease in the Pacific North-west, and it appeared for the first time in Western Oregon. This spread was correlated with a migration of the leafhopper from its breeding grounds east of the Cascade Range.

The virus seems to be confined to North America and possibly the Argentine. There is no authentic record of its presence in Europe, though a superficially similar disease has been described upon spinach in Trieste.

Control. The problem of the control of this very serious disease may be approached from the following three main standpoints : (1) Control of the beet leafhopper, (2) cultural practices, (3) the use of resistant varieties.

As regards the control of the leafhopper, direct attack on the insect with chemicals or mechanical means has not proved very effective in reducing the damage done by the disease. From the data obtained from ecological studies of the insect, however, and from past records of beet production, it is possible to predict the

probable insect populations in the spring and the period of migration in certain areas. Such predictions have been valuable in making it possible to avoid planting beets in years of heavy leafhopper attacks. Related to this phase of control is the study of the weeds or other plants which harbour the insect in its natural breeding grounds. A study of the plant successions in these breeding grounds has indicated the possibility of replacing introduced plants with the natural vegetation which is less favourable to leafhopper populations. For example, as a result of overgrazing on the foothill cattle ranges there has been much soil erosion and destruction of the natural vegetation such as sage brush (*Artemisia tridentata* Nutt.). This has been followed by an invasion of the Russian thistle, *Salsola kali*, harbouring the leafhopper (*Eutettix tenella*) (10). In certain districts, also, control of the leafhopper is claimed to have been achieved by the wholesale destruction of the Russian thistle (*Salsola kali*).

Of the cultural control methods the most important is timing the planting of the beets so as to get the crop advanced as far as possible before the spring influx of the beet leafhopper. In California, Idaho, and Utah such procedure has resulted in great benefit.

The use of resistant varieties for the control of plant virus diseases is very desirable whenever it is possible to produce such resistance. So far as the sugar-beet is concerned, some success has already been obtained with the curly-top resistant variety known as U.S. No. 1. This strain was derived from an extensive mass selection made from severely affected fields in Idaho and Utah during the curly-top epidemic of 1926. At the California Sugar Beet Conference on December 2nd, 1934, the yield from a planting near King City of this U.S. No. 1 beet was stated to amount to 18.6 tons with 20.8 per cent. sugar compared with 2.05 tons with 20.9 per cent. sugar from a susceptible variety. The increased yield from U.S. No. 1 seed used in the Grand Valley, Colorado, in 1934, is estimated at 2,828 tons of beets from 1,414 acres, and in the Delta district at 8,160 tons of beets from 2,245 acres. Two new varieties of curly-top resistant sugar beets have been recently produced, U.S. 38 and U.S. 34, and these will probably come into use on a large scale in the U.S.A. during 1937.

As far as the disease in the tomato in California is concerned, the development of resistant varieties of tomato may be the ultimate solution of the problem since there appears to be a definite though not very strong resistance in certain varieties. So far,

however, the greatest benefit has been obtained with temporary muslin tents which protect the plants from the insect invasion and create conditions less favourable for the development of the disease. With the summer crop this protection is of primary value during the first period of growth, or until about the end of June (61).

Host Range of Beta Virus 1

This host list is mainly compiled from the work of Severin and his colleagues.

Cruciferæ

- Armoracia rusticana.*
- Barbarea vulgaris.*
- Brassica alba.*
- B. oleracea acephala.*
- B. oleracea botrytis.*
- B. oleracea capitata.*
- B. rapa.*
- Matthiola incana.*
- Raphanus sativus.*

Violaceæ

- Viola tricolor*, var. *hortensis*.
- Viola cornuta*.

Chenopodiaceæ

- Beta maritima.*
- B. vulgaris.*
- B. vulgaris cicla.*
- Chenopodium murale.*
- Kochia scoparia*, var. *tricophila*.
- Spinacea olcracea.*

Geraniaceæ

- Pelargonium hortorum*.

Tropæolaceæ

- Tropæolum majus*.

Cucurbitaceæ

- Citrullus vulgaris.*
- Cucumis anguris.*

- C. melo cantalupensis.*
- C. melo inodorus.*
- C. melo reticulatus.*
- C. sativus.*
- Cucurbita maxima.*
- C. moschata.*
- C. pepo.*

Polygonaceæ

- Fagopyrum esculentum.*
- Rheum rhabonticum.*
- Rumex scutatus.*

Malvaceæ

- Hibiscus esculentus.*

Caryophyllaceæ

- Dianthus caryophyllus.*
- D. plumarius.*
- Stellaria media.*

Leguminosæ

- Cicer arietinum.*
- Medicago hispida.*
- M. sativa.*
- Melilotus alba.*
- M. indica.*
- Phaseolus lunatus.*
- P. vulgaris.*
- Trifolium hybridum.*
- T. incarnatum.*
- T. pratense.*
- T. pratense perenne.*
- T. repens.*
- Vicia atropurpurea.*
- V. faba.*
- V. sativa.*
- V. villosa.*
- Vigna sinensis.*

Umbelliferæ

- Anethum graveolens.*
- Anthriscus cerefolium.*

Apium graveolens.
Coriandrum sativum.
Foeniculum dulce.
Petroselinum hortense.

Dipsacaceæ

Scabiosa atropurpurea.

Amaranthaceæ

Celosia argentea, var. *cristata*.

Nyctaginaceæ

Mirabilis jalapa.

Compositæ

Coreopsis tinctoria.
Cosmos bipinnatus.
Helichrysum bracteatum.
Zinnia elegans.

Solanaceæ

Capsicum frutescens.
Lycopersicum esculentum.
Nicotiana rustica.
N. tabacum.
Solanum tuberosum.

Linaceæ

Linum usitatissimum.

Boraginaceæ

Borago officinalis.

Valerianaceæ

Valerianella locusta olitoria.

BETA VIRUS 2. Lind

Synonyms. *Beet Yellows Virus*, Prillieux and Delacoux, 1898.
Beet Mosaic Virus, Lind, 1915; *Sugar Beet Mosaic Virus*, ~~Rohlf's~~
 1921; *Sugar Beet Virus 2*, J. Johnson's Classification.

The Virus

Serological Reactions. Verplancke (69) has stated that he has obtained from hyper-immunised rabbits a specific antiserum with the sap of mosaic-diseased beets.

Thermal Death-point. The virus is inactivated by exposure for ten minutes to temperatures between 55° and 60° C.

Dilution End-point. The tolerance to dilution appears to be 1 : 1,000.

Resistance to Ageing. The longevity *in vitro* of the virus at a temperature of about 70° F. is between twenty-four and forty-eight hours.

Desiccation. There seems to be no exact information on this point, but it is doubtful if the virus will long resist desiccation.

Transmission. *Beta Virus 2* is transmissible by sap-inoculation if a fairly hard rubbing method is employed. It is not seed-transmitted. The insect vectors are the aphides *Myzus persicae* Sulz., *Aphis rumicis* and possibly *Macrosiphum gei* (see Chapter VIII).

Differential Host

Nicotiana tabacum. Tobacco. *Beta Virus 2* is not sap-transmissible to tobacco, but according to Hoggan (27) the virus can be transferred to this plant by the agency of the aphid *Myzus persicae* (see p. 538). No systemic infection follows, but local symptoms consisting of small, round, light brownish-yellow spots develop on the leaf lamina, 1 to 2 mm. in diameter, with very diffuse margins and often with a minute necrotic point in the centre.

Diseases Caused by Beta Virus 2

Chenopodiaceæ

Beta vulgaris. Sugar beet. *Sugar beet mosaic* (see Fig. 5).

The first sign of infection is the development, on one or more of the heart-leaves, of numerous small yellow spots, or irregular flecks, which increase in size. There may be a slight puckering of the leaf surface in the region of the spots. The next phase is the development on the young leaves of a bright mottling of very pale green or yellow against the darker green of the leaf. There may also be indentations of the leaf surface. The older green leaves exhibit a fine speck-like mottling of pale green on a darker background. Characteristic features are the bending back of the leaf near the tip, and curling and crinkling of the leaf margin, frequently followed by death of the tip. In more severe forms of the disease,



FIG. 5. *Beta Virus 2* (causing sugar beet mosaic). Mottling on young leaf of infected sugar beet.

the leaves are curled and distorted and the edges of the younger leaves often roll inwards, giving a tubular effect. The mottling also may be brighter. Growth is retarded and the plant is stunted and sometimes deformed. Under glasshouse conditions the first sign of infection is often a clearing of the veins whereby the veins of younger leaves are picked out in a lighter colour than the green background. There is no phloem necrosis or starch accumulation in the leaves. Affected beets mature somewhat earlier than healthy plants.

Histopathology. The external symptoms on the leaves are accompanied by internal changes in the anatomical structure. The palisade tissue is less differentiated from the mesophyll and the number of intercellular spaces is reduced. The nuclei of the cells are smaller, whereas the chloroplasts increase in size but are less brightly coloured. Intracellular bodies, or X-bodies, occur in some of the cells (46).

Other varieties of Beta vulgaris. Spinach beet, seakale beet, red or garden beet and mangold are all equally susceptible to infection with *Beta Virus 2*. The symptoms of the disease in the mangold are very similar to those in the sugar beet. The first sign of infection is the appearance of numbers of small light green flecks on the youngest leaves, or, more rarely, clearing of the veins. These symptoms are followed by the development of the characteristic mottling of the leaves. Similarly with garden beet, except that at first the symptoms may appear somewhat accentuated by contrast with the red background of the leaf.

Spinacia oleracea L. Spinach (see Fig. 7). The incubation period of the disease in spinach varies greatly according to the time of year and the prevailing environmental conditions. The minimum time appears to be about six days, but the period may be as long as three weeks. The onset of symptoms is quite sudden, the first sign of infection being usually a marked downward arching of the young leaves accompanied, or soon followed, by the appearance of numerous bright golden yellow flecks on the young leaves. These flecks are round or irregular in form, less than 1 mm. in diameter, and often with a minute darker point in the centre. Frequently they are so numerous as to coalesce, forming large yellow areas. There is no faint uniform chlorosis of the foliage at this stage as is found in spinach plants affected with *Cucumis Virus 1*, which causes *spinach blight* (see p. 56). As the disease progresses the yellow coloration gradually spreads over the foliage, the plant becomes much stunted and growth soon ceases.

Necrosis of the outer leaves then develops, the leaves usually dying back from the tip. The necrosis spreads slowly inwards until the plant is reduced to a central whorl of small mottled and puckered leaves; at this stage the plant usually dies. The disease produced in spinach by *Beta Virus 2* differs from that produced in the same plant by *Cucumis Virus 1 (spinach blight)* in the occurrence of the fine discrete spotting as an early symptom of infection with the former virus and in the absence of any marked malformation of the leaves of seedling plants as is the case with infection by the latter (27).

Perpetual Spinach. The symptoms on this variety are less severe than on the annual plant and consist for the most part of a mosaic mottling of small light yellow spots or flecks on the darker green of the leaf. This plant, however, is of considerable importance in relation to the disease since it affords the virus an opportunity for overwintering.

Chenopodium album Linn. White goosefoot. This common weed is susceptible to infection with *Beta Virus 2*, and may play a certain part in the spread of the disease. When experimentally infected, symptoms develop in about seven days in the form of circular zones with an indistinct central spot. Later, symptoms of general infection appear on the young leaves. Isolated yellow spots develop together with a curling-under of the leaf edges; such affected leaves finally shrivel and die off.

Certain other weeds such as *Amaranthus retroflexus* and *Sonchus arvensis* are said to be susceptible to infection with this virus.

Geographical Distribution. The mosaic disease of beets was first noted in 1898 on garden beets in Northern France and in the vicinity of Paris. Reports on this disease have been made in Denmark, in Germany and in Sweden on sugar beet and garden beet. The disease was first reported in the United States of America in 1915, having been observed on sugar beets in Northern Colorado and Western Nebraska. The Plant Disease Reporter of the United States Department of Agriculture records the occurrence of sugar beet mosaic in Utah and New Mexico in 1927, Utah and Texas in 1928, and Washington in 1929 (31). The disease is now present in England and in all the sugar beet growing districts of Europe.

Control. The only methods of controlling beet mosaic, at present known, consist in efforts to keep down such weeds as docks, white goosefoot, poppies and sowthistle, which may serve, in certain instances, both as sources of virus infection and as hosts for

the insect vector *Aphis rumicis*. Mosaic-infected beet or mangolds should be removed as soon as possible. It should also be remembered that second-year beets which are being grown for seed, and certain biennial plants like seakale beet and spinach beet are very frequently infected with the virus. Such plants are therefore a common source of infection for crops of sugar beet or mangolds growing in their vicinity. According to Böning (7) the symptoms of the disease are most severe if there is a deficiency of phosphoric acid ; this therefore should be avoided. No varieties of sugar beet or mangolds resistant to mosaic are known.

BETA VIRUS 3. Wille

Synonyms. *Sugar Beet Virus 3*, J. Johnson's Classification ; *Sugar Beet Leaf-curl Virus*, Wille, 1928 ; *Sugar Beet Leaf-crinkle Virus*, Wille, 1928.

The Virus and its Transmission. The virus is apparently not sap-inoculable and there is no information on its properties. It does not appear to be seed-transmitted. The specific insect vector is a Tingid bug, *Piesma quadrata* Fieb. (see p. 469). According to Kaupmann the larval stages of this insect are unable to transmit the virus (compare the insect vectors of *Callistephus Virus 1* and *Lycopersicum Virus 3*, pp. 215, 297). Adult insects, however, after having once fed on affected beet, seem capable of infecting healthy beet for the remainder of their lives. The virus is said to overwinter in the insect which hibernates in grassy ditches and other places.

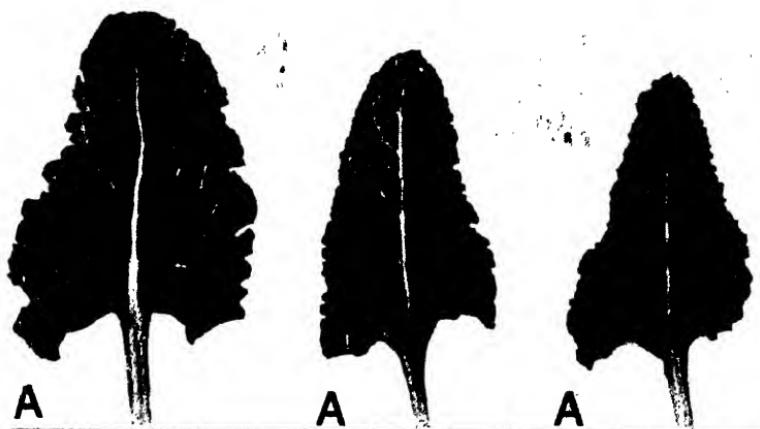
Disease Caused by Beta Virus 3

Chenopodiaceæ

Beta vulgaris. Sugar beet. *Crinkle* (Figs. 6, A, 6, B). The disease caused by this virus is known in England as crinkle, and in some parts of Germany as "Kopfsalat." The earliest symptom to

FIG. 6.

- A. Leaves of sugar beet affected with *Beta Virus 3* (crinkle), showing clearing of the veins.
- B. Sugar beet plants in the field, affected with *Beta Virus 3*; *d*, diseased; *h*, healthy.
- C. Plants of *Anemone nemorosa* affected with *Anemone Virus 1* (alloiophylly). (A and B, after Nitsche; C, after Klebahn.)



appear is a glassy or translucent appearance of the veins of the leaves. Later, the veins become swollen and since they do not grow so fast as the rest of the leaf, the leaves acquire a markedly crinkled appearance. The crown is stimulated to increased activity and forms new leaves which remain small and curve inwards to form a compact bunch; this gives the top the general appearance of a head of cabbage lettuce (46). The incubation period of the disease in the plant is from twenty-one to sixty-five days. The main symptoms of the disease are swelling, crookedness, and glassiness of the leaf veins and petioles, crinkle due to inflation of the leaf parenchyma, arrest of growth, dying of the older leaves and death before the harvest. The disease develops most rapidly if the bugs are numerous, feeding prolonged and temperature high. Some beets are immune, but the immunity is peculiar to individual plants and is not a varietal character (75). Three distinct forms of the disease have been recognised, a severe and progressive form starting early in the year, a similarly severe form interrupted by periods of normal growth and a slight form starting much later. It is not known whether these forms of the disease are due to different strains of the virus.

Host Range. Wild chenopodiaceous plants are not susceptible to infection, but beans (*Phaseolus*) have been infected artificially (75). It is possible that spinach is also susceptible.

Geographical Distribution. At present the virus appears to be restricted to certain parts of Germany and Poland. In Germany it is chiefly confined to two districts, Anhalt and Silesia. In the region lying between these two districts there are also small areas where the disease is prevalent. In the provinces of Baden, Bavaria, Schleswig-Holstein, East Prussia and Pomerania, crinkle has never been seen (46).

Control. The only available methods of control at present are those directed against the insect vector, *P. quadrata*. Insecticides have proved ineffective or too expensive and cultural methods are the best. The method which has been found most satisfactory is to sow the seed early. When the bugs are feeding on the first crop it is ploughed under and a further sowing is made. This method is employed in the neighbourhood of Dessau, in Anhalt. In Silesia a cheaper method of trap-sowing is recommended; this consists in sowing strips of beet round the edges of the field and ploughing them under before the beet on the rest of the field is showing through. The trap strips should be sown some 8 to 6 metres from the edge of the field and the strips should be

6 to 9 metres wide. The object of leaving a bare strip round the edges of the field is to prevent the bugs returning to their winter quarters in cold and damp weather. The corners of the field are not sown, and a space 3 to 5 metres long next to the corners is left undrilled. This is necessary so that the ploughed-in bugs at the corner are not dug up in turning. The trap strips are drilled about the second week in April (earlier sowing is not so good, as it is the very young beet which is the most attractive to the insect). The beet is drilled much closer than for an ordinary crop, the distance between the rows being 7 to 8 inches. It is then left unthinned until it is ploughed under. The main crop is sown before the trap crop is ploughed under and this should be done about a week after the finding of the first eggs. The ploughing of the trap strip should be carried out about two days before the main crop appears through the ground. This time usually corresponds with the flowering of the horse chestnut. The ploughing must be done in the early morning before the sun rises as in the middle of the day the bugs are active and flying. The ploughing should be about 8 inches deep if possible, and the soil must be completely inverted. Immediately afterwards the land should be rolled. The strip can then be harrowed and sown with beet (46).

BETA VIRUS 4. Roland and Quanjer

Synonyms. *Jaunisse Virus*, Roland; *Vergelingsziekte Virus*, Quanjer; *Yellows Virus*, Petherbridge and Stirrup.

The Virus and its Transmission. There is at present no information on the properties of this virus. The virus is not sap-transmissible nor is it carried in the seed. The insect vectors are the same as for *Beet Virus 2*, i.e., *Myzus persicae* Sulz and *Aphis rumicis* (= *A. fabae*).

Disease Caused by Beta Virus 4

Chenopodiaceæ

Beta vulgaris. Sugar beet. *Virus yellows* (see Fig. 7). The symptoms of virus yellows are characteristic. They do not generally appear until about mid-season. The young heart leaves are not usually affected, and this is a point of difference from the infection caused by *Beta Virus 2*, but the older, outer leaves change in colour, first to a yellowish-green and then to a distinct yellow. Such symptoms are common also to other forms of yellowing not

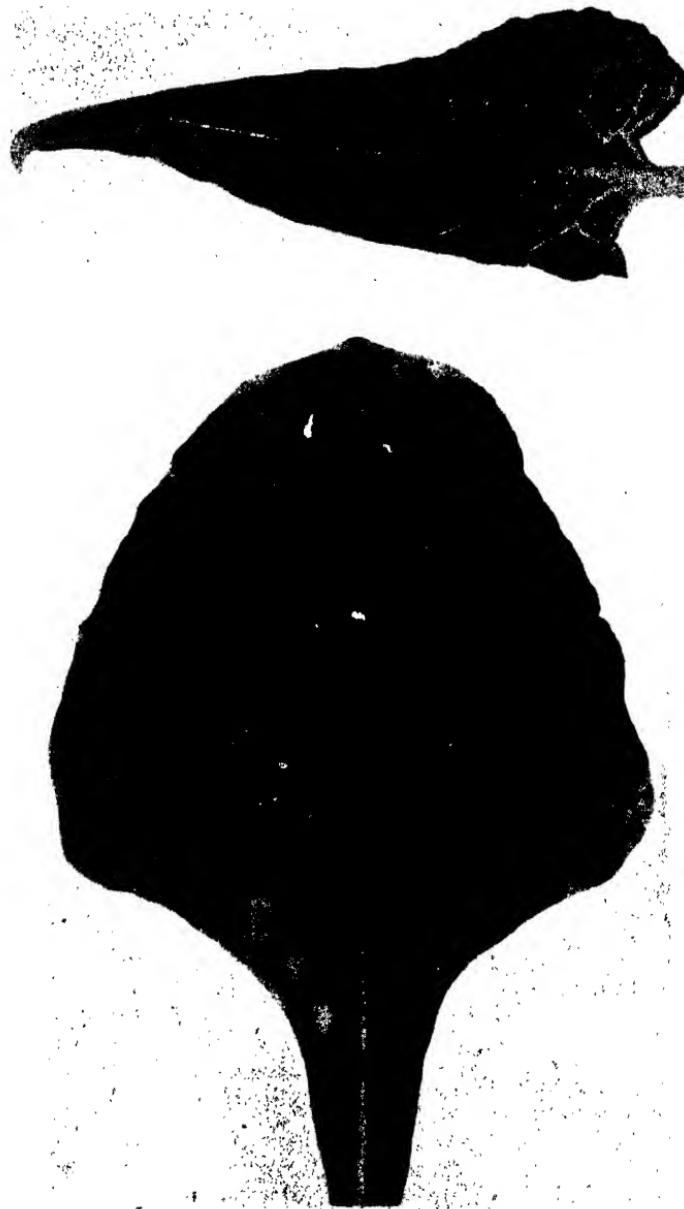


FIG. 7.

(Top) Leaf of spinach infected with *Beta Virus 2* (sugar beet mosaic virus).
(Bottom) Leaf of sugar beet infected with *Beta Virus 4* (virus yellows).
(Bottom after Quanjer.)

due to virus infection, but in virus yellows the texture of the leaves alters, and they become thick and very brittle. When walking through a crop badly attacked by virus yellows an unusual rustling or crackling noise may be heard, caused by the knocking together or breaking of the thick brittle leaves of the diseased plants (46). The chief characteristics, then, of this disease are : yellowing, brittleness, shortness and thickness of the nearly full-grown leaves, gummosis in the sieve tubes throughout the plant, except in its youngest parts, and accumulation of starch in the leaves (53, 54). The host range of *Beta Virus 4* is not known ; at Wageningen, in Holland, the virus has been found overwintering in the roots of winter-resistant strains of *Beta maritima* (55).

Geographical Distribution. *Beta Virus 4* has been recorded from the British Isles, Germany, Holland and Denmark, and is probably present in North America.

BETA VIRUS 5. Coons, Kotila and Stewart

Synonym. *Sugar Beet Savoy Disease Virus*, Coons, Kotila and Stewart, 1937.

The Virus and its Transmission. Nothing is known at present of the properties of this virus ; it does not seem to be sap-transmissible. The insect vector is a Tingid bug, *Piesma cinerea*. The virus appears to overwinter in the vector in the same way as *Beta Virus 3*.

Diseases Caused by Beta Virus 5

Chenopodiaceæ

Beta vulgaris. Sugar beet, garden beet ; *Savoy disease*.

Affected plants show dwarfed, down-curled, savoyed leaves, the most pronounced effects being found in the innermost leaves. The primary symptoms are veinlet-clearing, followed by the thickening of the veinlets, giving the dorsal leaf surface a netted appearance. The roots of affected plants, in late stages, show phloem necrosis and flesh discolouration which simulate the effects of curly-top disease (*Beta Virus 1*). The incubation period of the virus in sugar beet is from three to four weeks.

Geographical Distribution. The virus has only been recorded from the United States of America, where it has been found in Michigan, Ohio, Minnesota, Nebraska, South Dakota, Colorado and Wyoming (14).

PELARGONIUM VIRUS 1. Pape

Synonym. *Pelargonium Leaf-curl Virus*, 1927.

The Virus and its Transmission. Very little is known concerning this virus, it is not apparently sap-inoculable, at all events between Pelargoniums, although Verplancke (68) states that inoculation



FIG. 8. *Pelargonium Virus 1* (*Pelargonium leaf-curl virus*). Note the yellow star-like spots and leaf distortion.

to tobacco produced diffuse discoloured lesions on the inoculated leaves. The writer, however, has been unable to produce any lesions in tobacco other than those which appeared to be due to a toxic effect of the Pelargonium sap. The virus has been transmitted by grafting; the insect vectors are not definitely known, though there is some evidence which suggests that the aphid *Macrosiphum pelargonii* (see p. 585) may be the vector.

Disease Caused by Pelargonium Virus 1**Geraniaceæ**

Pelargonium zonale. The name "leaf-curl" given to the disease is not very descriptive of the symptoms. The young leaves first exhibit pale chlorotic spots which gradually develop into rounded or more or less stellate or dendritic blotches of a bright yellow colour. Occasionally a clearing of the finer veins of the young leaves may be observed. Often the spots show a deep yellow centre surrounded by one or more concentric halos of a lighter colour. Later, necrosis of the spots sets in and the tissue dries up and becomes brown. Growth in these areas ceases, but it is continued in the surrounding tissues of the lamina with the result that the leaf develops a crinkled and puckered aspect, sometimes accompanied by splitting (see Fig. 8). In severe cases the whole plant becomes malformed and degenerate. Certain plants exhibit only livid, translucent, oily, circular spots up to 4 to 6 mm. in diameter, the centre of which is a normal green surrounded by a paler zone. Both *Pelargonium zonale* and *P. hederaceum* exhibit these symptoms.

The symptoms appear first in the spring in cuttings taken from plants of the previous year, and one rather striking feature of the disease is the apparent recovery which sets in. As the season progresses the new leaves which develop do not show the symptoms described, and since the older affected leaves are cast off, the plant may then appear quite healthy. Cuttings taken in the autumn from seemingly healthy plants, however, exhibit the symptoms again in the new leaves that develop in the spring, showing that the virus is still present. Plants grown under hot, dry conditions generally show the symptoms more intensely than those grown in cooler and moist surroundings (47).

Histopathology. Verplancke (68) gives some details of the histological changes caused by the disease, in the oily spots the palisade parenchyma is not differentiated and there are no lacunæ in the spongy parenchyma. The tissue of the dendritic or star-shaped lesions also consists of a parenchyma of undifferentiated cells.

Geographical Distribution. The disease has been described by Pape in Germany (45) and by Marchal (39) and Verplancke (68) in Belgium. In England the disease has been recorded from the Birmingham district, from Leeds, Reading, Rugby, Cambridge and London. It is therefore widely distributed in England.

Control. Little can be written at present concerning any

method of controlling the spread of this disease. In England the trouble has been seen chiefly in the varieties Paul Crampel and King of Denmark. Pape considers that zonal Pelargoniums with light salmon-coloured flowers as well as *Pelargonium peltatum* and "Edel" varieties are more resistant than others. He recommends cutting back the plants and spraying with sulphur-containing materials. Pape further recommends repotting or transplanting to the open after the removal of the affected leaves. Badly affected plants are best destroyed by burning, and cuttings should on no account be taken from them. Shading and keeping a moist atmosphere during periods of hot weather will help to suppress the symptoms. Fumigation or spraying to keep aphides and other insects in abeyance is a wise precaution.

PASSIFLORA VIRUS 1. Noble

Synonyms. *Passion Fruit Woodiness Virus*, Cobb, 1901; *Passion Fruit Bullet Disease Virus*.

The Virus and its Transmission. There is no information at present on the properties of the virus. It is sap-inoculable, the method used by Noble (43) being the insertion in the vascular system of some cotton wool soaked in virus sap. The natural means of spread of the virus are not known. It is possible that a certain amount of dissemination may take place during pruning operations.

Disease Caused by Passiflora Virus 1

Passifloraceæ

Passiflora edulis, Sims. Passion fruit. The foliage of vines affected with the woodiness disease is abnormal. Such vines have a general appearance of unthriftiness and appear as if suddenly checked in growth. The leaves of the terminal shoots may be stunted and are frequently curled, twisted and deformed. Changes may occur in the chlorophyll-bearing tissues which result in the development of a yellowish-green chlorosis, or there may be formed a definite mosaic of abnormally light green and dark green areas on the leaf. The tissues of the leaf between the veins may be raised or sunken, thus giving the leaf a puckered or crinkled appearance. Light yellowish-green spots may develop on older leaves which previously were full-grown and otherwise quite healthy in appearance. The stems of affected plants, particularly in the region of terminal shoots, may develop mottled dark green

areas which are in marked contrast to the normal green coloration of healthy plants. These foliar symptoms have been observed under field conditions both in seedlings and in old vines. There is some evidence that symptoms are masked under high temperature conditions.

Fruits which are ripened on normal vines are 'dark purple in colour, somewhat ovoid in shape and are generally symmetrical in appearance. "Woody" fruits, on the other hand, are generally misshapen and deformed. Such fruits are often undersized and when not obviously malformed may be somewhat spherical. This symptom has given rise to the alternative name of "bullet disease." The surface of the fruit may be smooth as in the case of normal fruits, but more generally it is characterised by the development of irregularly-shaped areas of tissue which appear to have burst through the skin of the fruit. Diseased fruits are characteristically hardened, offer considerable resistance to pressure, and, in contrast to normal fruits, are not readily cut through. When they are cut through, the tissues of the pericarp or rind will be noticed to be abnormally thickened (48).

Histopathology. Histological studies of the pericarp of abnormal fruits indicate that it differs materially from that of a normal fruit. Extensive changes occur in the tissues which constitute the innermost section of the pericarp. The cell walls are thickened and pitted, and the cells are either devoid of, or almost devoid of, their normal contents. Tests indicate that these cells are strongly lignified. The lignification may be restricted to cells adjacent to the hypodermal layer of sclerenchymatous tissue, or it may extend throughout the whole of the inner portion of the pericarp.

Host Range. *Passiflora cœrulea* L. has also been found infected with *Passiflora Virus 1*.

Geographical Distribution. Woodiness of passion fruit has long been known in New South Wales where it causes serious damage. The disease also occurs throughout Australia. Bewley records a mosaic disease of passion flower in England in 1923, and the writer has observed a mosaic disease, also in England, on *P. cœrulea* L.

Control. Noble (48) suggests the following methods of control for the disease :

- (1) Seedlings should not be raised in proximity to diseased vines.
- (2) Only healthy seedlings should be planted out. Symptoms of woodiness disease are readily detected in seedlings in the spring,

and any diseased plants should be immediately removed and destroyed.

(3) Careful systematic inspections should be made of the vines in young plantations, and any which show abnormality of growth should be destroyed.

(4) Very careful inspection should be made of the vines at the time of pruning, particularly in the first season of growth. It is quite possible that infection can be carried on the hands or pruning knife, so that these should be well washed after a diseased vine has been dealt with and before a healthy vine is touched.

(5) In plantations more than a year old, the vines have become entangled and removal of an affected plant under these conditions involves risk of contamination to neighbouring healthy vines. It is therefore better to cut through the roots of the diseased plant and allow it to dry up *in situ*. It can then be removed later with impunity.

(6) It is usually not practicable to spray the vines to keep down possible insect vectors, but it may be worth while to remove all weeds which are likely to harbour such insects.

CUCUMIS VIRUS 1. Doolittle

With *Cucumis Virus 1* commences the discussion of the viruses primarily associated with the cucumber and other members of the Cucurbitaceæ. There exists a certain amount of uncertainty concerning the nomenclature and differentiation of this group of viruses and these difficulties are increased by the occurrence of numerous strains which resemble the type virus in certain characteristics, notably in their physical properties, but may differ markedly in their symptomatology. Ainsworth (1) differentiates three viruses causing cucumber mosaic in England, and James Johnson in his proposed system of virus nomenclature considers that there are altogether five basic mosaic viruses with thirteen strains in addition. Twelve of these strains are variants of one virus, *Cucumis Virus 1*, while the thirteenth strain is considered to be allied to *Cucumis Virus 2*. In this account the basic or type viruses are limited to two.

Synonyms. *Cucumber Mosaic Virus*, Doolittle, 1920; *Common Cucumber Mosaic Virus*, *Cucumber Yellow Mosaic Virus*, Ainsworth, 1934; *Cucumber Yellow-mottle Mosaic Virus*, Ainsworth, 1935; *Cucumber White Pickle Mosaic Virus*, Gilbert, 1916; *Tobacco Puff Virus*, Valleau and Johnson, 1928; *Spinach-*

blight Virus, McClintock, 1918; *Southern Celery Mosaic Virus*, Wellman, 1935; *Celery Virus 1*, Wellman, 1935.

The Virus

Like *Nicotiana Virus 1*, this virus exists in a number of allied strains, some of which produce symptoms very different from those characteristic of the type virus. It is not, however, practicable to give detailed descriptions of all these strains, so that attention is mainly confined to the type virus and those strains which are of particular interest and importance.

Resistance to Chemicals. The virus is inactivated by 50 per cent. alcohol in one hour, by 0·5 per cent. solutions of formaldehyde, phenol, and copper sulphate and by mercuric chloride in a strength of 1 : 2,000.

Serological Reactions. An antiserum can be obtained from hyper-immunised rabbits which neutralises the virus. The anti-serum is specific in its reaction and inactivates only this virus and its strains. This reaction can be used to show that a yellow strain is closely related to a green strain in spite of the differences between their respective symptoms. It also shows that the yellow strain is wholly unrelated to a superficially indistinguishable yellow strain of *Nicotiana Virus 1* (12).

Thermal Death-point. The virus is inactivated by exposure to temperatures between 60° and 70° C. Ainsworth (1) gives the inactivation point as 60° C. for ten-minute exposures, and Doolittle (17) gives it as 70° C. for similar exposures.

Dilution End-point. This is found to be 1 : 10,000.

Resistance to Ageing. The longevity *in vitro* is between seventy-two and ninety-six hours at room temperatures.

Desiccation. The virus is destroyed by drying.

Filterability. The virus is filterable through a Berkefeld (normal) filter, but not apparently through Pasteur-Chamberland filters. The inability of the virus to pass these filter candles is probably due to its great capacity for adsorption. Passage of a virus-sap suspension through a sand-and-pulp filter or a bed of Fuller's earth renders the filtrate non-infectious because of the adsorption of the virus. This property is also possessed by *Solanum Virus 2* (Potato virus Y), and there seems to be a certain affinity between these two viruses. *Cucumis Virus 1* can be eluted by changing the pH of the suspension to the acid side, about pH 6 to 6·7 (66).

Transmission. The virus is easily transmissible by the sap.

The question of seed transmission of this virus is of great practical importance, but opinion is divided on the subject. According to Doolittle and Walker (19) the virus is transmitted through the seed of the wild cucumber (*Micrampelis lobata*), but not through the seed of the cultivated cucumber. They say (*loc. cit.*, p. 56), "Further trials with the seed of mosaic cucumber, squash, muskmelon and pumpkin plants indicate that seed transmission of the disease probably does not occur or is so rare as to be of little significance." On the other hand, Bewley considers that the virus is seed-borne and that the use of clean seed is an important measure in the control of the disease; he says, "It seems reasonable to conclude that seeds taken from fruits borne by mosaic-infected plants do not invariably produce infected progeny, neither do they invariably produce healthy progeny, for a small percentage of infected seedlings occurs from time to time" (6). The matter is, therefore, not proven so far as the cultivated cucumber is concerned, and in this connection it is well to bear in mind the wide host range of the virus and the many possible sources of infection from which young cucumber seedlings might become contaminated. Mahoney (38) offers evidence that the virus is carried in the seed of certain inbred lines of muskmelon, *Cucumis melo* L. He finds that in the spring of 1932 and 1933 several cases of mosaic occurred among seedlings grown from muskmelons in Michigan in a form strongly suggestive of seed transmission. Selection was made from these plants, and in every case where the plant was infected it transmitted the virus through the seed, the average percentage transmission being 15·6. The virus was also found to be seed-borne in a number of commercial varieties of muskmelon, the average percentage of infection ranging from 8 to 27 per cent.

As regards insect transmission, there appear to be several vectors. The following aphides are said to transmit the virus, *Myzus persicae* Sulz., *M. pseudosolani*, *M. circumflexus*, *Macrosiphum gei* (= *solanifolii*) (26), and *Aphis gossypii* (17, 19). Doolittle considers that the leaf-eating beetles *Diabrotica vittata* and *D. duodecimpunctata* are the vectors of the virus under field conditions in America. Information on the aphides mentioned will be found in Chapter VIII.

Differential Host

On *Datura Stramonium* symptoms develop five to seven days after inoculation at a mean daily temperature of 60° F. Pale spots develop on the inoculated leaves, and these are followed about

two days later by a mosaic mottle together with the formation of characteristic chlorotic ring and line patterns. The rings, which are never necrotic, are of a darker green than the background and are frequently concentric.

Diseases Caused by *Cucumis Virus 1*

Ranunculaceæ

Delphinium. This plant is very susceptible to the virus and is frequently found infected in English gardens. Affected plants present a chlorotic appearance, and there are pale areas on the leaves usually following the veins. A rather faint green mosaic mottle is often present. Annual plants of larkspur (*Delphinium consolida* L.) when naturally infected in the field show marked stunting and chlorosis and rarely produce flowers. Diseased plants are much flattened in growth and have a rosetted appearance, leaflets are chlorotic, downwardly curled and sometimes mottled with light green.

Aquilegia sp. Columbine. This garden plant is occasionally found naturally infected with *Cucumber Virus 1*; the leaves show clearing of the veins followed by a mosaic mottle. Necrosis of the older leaves is sometimes present and the plant is stunted. Flowers are very few or entirely absent.

Cruciferæ

Nasturtium officinale. Water-cress. Affected plants show a distinct yellow mottling of the leaves which are slightly deformed. The plants are somewhat stunted (2).

Violaceæ

Viola cornuta. *Viola*. Violas in gardens in the Cambridge district have been observed by the writer to be affected with this virus. This disease shows itself chiefly in the flower petals. Such flowers do not have an abrupt change from one colour to another, but exhibit a smoky or smudged appearance with a characteristic pencilling or flecking of the petals. The leaves of affected plants are slightly curled and chlorotic, especially near the tips. On the whole the effects of the disease on the health of the plant are not very severe.

Polygonaceæ

Fagopyrum esculentum Gaertn. Buckwheat. Affected plants exhibit a severe systemic mottling on the leaves together with malformation and a general stunting.

Phytolaccaceæ

Phytolacca americana (decandra). Pokeweed. Common pokeweed. The disease first appears on the young leaves as a mottling of light greenish-yellow in which the light areas are of small size. This mottling is accompanied by a downward curling of the midrib of the leaf, similar to that found in cucumber plants affected with the same virus. The light green portions of the leaf may later include the greater part of its surface. They are irregular in outline and do not appear to be delimited by the veins of the leaf. In the later stages of the disease the plants have a typically mosaic appearance with wrinkled and blistered leaves (19).

Chenopodiaceæ

Spinacia oleracea. Spinach. *Spinach blight*. The disease caused by this virus in spinach is known as "spinach blight," and the first recognisable symptom consists of a faint general chlorosis sometimes involving the entire foliage, sometimes, especially with older plants, apparent only on the younger leaves. As growth continues the chlorosis becomes more intense and spreads outward until the whole plant becomes yellow. At the same time, newly developing leaves begin to show malformation in varying degrees. In mild cases the malformation consists in a reduction in width of the lamina, frequently accompanied by considerable wrinkling and an inward rolling of the leaf margin. In more severe cases some leaves become reduced to a fine ribbon ; others are puckered, twisted and distorted ; while some develop a mottling with marked savoying of the darker areas. Later a progressive necrosis develops, involving first the outermost whorl and slowly working inwards. As the affected leaves turn brown and shrivel, growth declines and the plant is gradually reduced in size until only a small central cluster of yellow malformed leaves remains. Death usually ensues at this stage (27). McClintock and Smith (41) divide the course of the disease into eight arbitrary stages, which may be summarised as follows :

- (1) A very slight yellowing of the younger leaves of infected plants and occasionally of one or more older leaves.
- (2) A progressive yellowing of the younger leaves.
- (3) Malformation of the younger leaves which become much wrinkled, narrowed and mottled ; yellowing of the older leaves and some stunting of the plant.
- (4) Distinct stunting of the plant with cessation of growth ;

yellowing of the entire foliage; mottling of the older leaves and extreme malformation of the younger ones which are finely savoyed and feathery in appearance.

(5) Browning and death of parts of the older leaves, usually progressing from the outer tips inwards.

(6) Total disintegration of the older leaves and some browning of the central ones.

(7) Browning of the younger leaves.

(8) Death of the plant which has become reduced to a very small central whorl of leaves.

Amaranthus retroflexus. Pigweed. Affected *Amaranthus* plants are much dwarfed and usually reach a height of only 8 to 10 inches, while healthy plants are often 2 to 3 feet tall. The diseased plants show a typical mosaic mottling of the foliage, the major portion of the leaf becoming yellowish-green with smaller dark green areas scattered over the surface. The petioles of such leaves are usually longer in proportion to the blade than those of healthy plants, and this, together with the shortening of the internodes, gives the mosaic plants an abnormally spreading habit. The leaves of mosaic plants are usually smaller, narrower, and more irregular in outline than those of healthy plants. Mosaic plants usually develop only a single stem and do not branch as commonly as do healthy pigweeds. Seed is produced by diseased plants, but only in small amounts (19).

Geraniaceæ

Pelargonium hortorum Bailey. Bedding geranium. Affected plants of this species exhibit a mottling of the leaves together with a stunting of the whole plant. It has not been found possible to infect this plant by sap-inoculation with the virus, but only by means of the insect vector (*Aphis gossypii*) (see p. 515).

Geranium carolinianum L. Crane's-bill. Symptoms consist largely of stunting, mottling and some distortion of the leaves. Inoculation by means of aphides produces the disease, but rubbing methods fail to transmit the virus. Systemic symptoms are quite severe, but do not inhibit seed production (74).

Tropæolaceæ

Tropæolum majus. This plant is susceptible to experimental infection with *Cucumis Virus 1*, which produces diffuse local lesions on the inoculated leaves without any definite systemic symptoms.

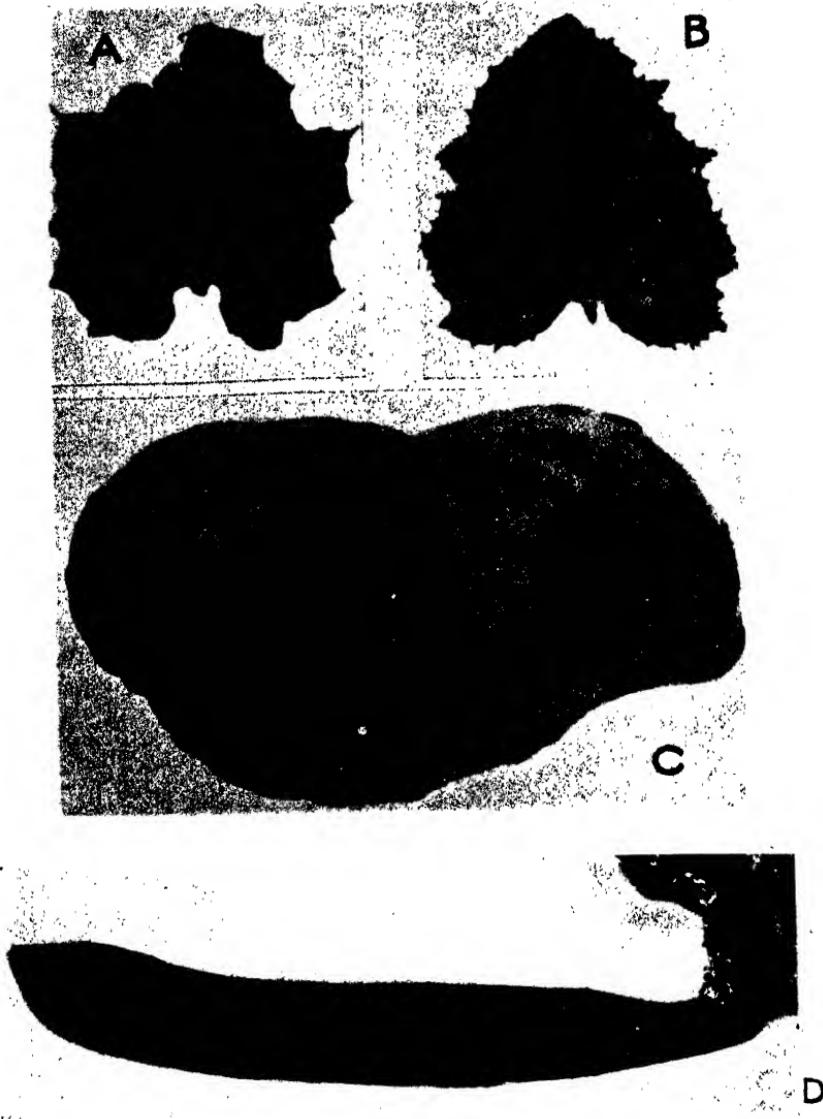


FIG. 9. *Cucumis Virus 1* (cucumber mosaic virus).

- A. Mosaic leaf from a *Lagenaria* gourd.
- B. Mosaic leaf from a cantaloupe, natural infection.
- C. Mosaic fruit of pumpkin, showing large dark green swellings on a yellow background.
- D. Fruit from infected cucumber, showing marks caused by the virus.
(A-C, after Doolittle; D, after Ainsworth.)

Cucurbitaceæ

Cucumis sativus. Cucumber. The period of most general infection begins when the plants are about six weeks old and growing vigorously. At this time they have six to eight leaves and are commencing to run. The first symptoms in all cases appear in the young leaves which develop small greenish-yellow areas, often not more than a millimetre or two in diameter, occasionally circular, but more often limited in outline by the smaller veins of the leaf. These spots are slightly more translucent than the remainder of the leaf and are often scarcely visible except by transmitted light. The characteristic symptoms are a yellow mottle on all leaves developed after infection, some leaf distortion and stunting of the plant. Less commonly the normal green of the leaf changes to a peculiar yellow at the tip and the sharply defined yellow areas do not appear. Accompanying these symptoms there is a gradual downward curling of the edges of the leaf, and the surface presents a finely wrinkled appearance, the tissue between the small veins becoming slightly raised so as to form minute convex surfaces. Affected leaves are wrinkled and savoyed in appearance and may be somewhat distorted and curled. All growth subsequent to infection is much dwarfed, the stem internodes are shortened, the leaves attain only about one half normal size, and the petioles are reduced in length. Plants infected at an early stage blossom sparingly and set few fruits. They send out few runners and have a bunched and bushy habit of growth, with the leaves lying close to the ground in a rosette-like clump.

The symptoms on the fruits of the cucumber are usually well marked and assist the identification of the disease. The stem-end of the young fruit first becomes mottled with yellowish-green, and this gradually spreads over the entire fruit. As this progresses the body of the fruit ordinarily becomes a light yellowish-green, intermingled with spots of a much darker green colour. These dark portions are usually raised above the surrounding surface in such a way as to form wart-like projections and often produce distortion of the fruit (see Fig. 9, D). Occasionally in the later stages of the disease, fruits are produced which are of a smooth greenish-white colour and somewhat misshapen with irregular green areas. These white fruits are responsible for the name "white pickle," which was the term originally applied to the disease in some parts of the U.S.A.

The flowers of affected cucumber plants are not streaked or

variegated, as are those of many plants infected with mosaic viruses (see pp. 11 & 411), and this is interesting in view of the fact that a strain of this virus does produce flower variegations in certain plants (see *Primula obconica*, p. 68). The flowers are, however, reduced in number and when produced at a late stage of the disease are frequently dwarfed (17).

Cucumis melo L. Melon, muskmelon. First signs of infection appear on the youngest leaves which turn light yellow in colour and develop a sharp downward curve. Later all the younger leaves of the plant develop a typical mosaic mottle. The light yellow portions of the leaf are of irregular outline and about the same size and colour as the spots on the young leaves of the cucumber. The dark areas are more definitely outlined on the melon and the leaves show a very pronounced curling. The characteristic symptoms found on the older leaves of the cucumber are less common on the melon. The older leaves gradually become yellow, but show little of the tendency to die early which appears in mosaic cucumber leaves of the same age. The young fruits of mosaic melon plants are often mottled and a few dark green warts may appear, but as the fruits mature the symptoms of the disease become less pronounced and the fruits are nearly normal in appearance (17).

Cucurbita pepo. Vegetable marrow, pumpkin. Doolittle in America (17) describes this disease on *C. pepo* as an extreme mottling and wrinkling of the younger leaves, the general colour being a lighter yellow than normal. The older leaves often show a rapid yellowing and wilting corresponding to that found on cucumbers in the greenhouse. The fruit may be mottled and deformed, but few fruits develop on an infected plant. A mosaic of vegetable marrow is common in England, and was first recorded by Ogilvie *et al.* in 1928 (44), who describes the symptoms as a very marked mottling of the leaves, taking the form of pale yellow markings which tend to run in wavy lines or circles. The leaves also tend to become puckered. Affected shoots have decidedly shortened internodes and a tendency to branch. The diseased fruits are mottled and covered with somewhat circular wart-like areas. All the young leaves and shoots of an infected plant ultimately show the symptoms, but the older leaves remain apparently normal in appearance. Much of the young fruit dries off without developing (see Fig. 9, C).

There seems little doubt that the same virus or a strain of the same virus affects vegetable marrows both in America and



A



B

FIG. 10. *Cucumis Virus 1* (cucumber mosaic virus).

A. Cucumber plant, showing the mosaic mottling caused by this virus. This plant was inoculated from an infected *Aquilegia*.
B. Infected plant of *N. glutinosa*, showing systemic symptoms.

Europe, but there is still uncertainty whether the virus causing mosaic of vegetable marrow is *Cucumis Virus 1* or a strain of the type virus.

Cucurbita maxima Duchesne. *C. pepo*, var. *condensa*. Squashes. Younger leaves develop an extremely savoyed appearance, the darker parts of the leaf being much more definitely raised above the leaf surface than in the case of the cucumber. The light spots on the leaf are pale yellowish-green and tend to coalesce and form a few large blotches rather than the smaller and more numerous spots found on the cucumber. The most marked symptoms have been found on the Summer Crookneck and Cocozelle bush varieties, while those of the Hubbard type show less severe symptoms.

Fruit symptoms on the Summer Crookneck squash are characteristic, the fruits are distinctly mottled and the warty character is even more pronounced than on the cucumber. The symptoms differ from those on most other cucurbits, in that the raised portions of the fruit are lighter in colour than the surrounding surface. The contrast is often very pronounced, the warts being bright orange-yellow and the remainder dark green.

Citrullus vulgaris Schrad. Watermelon. Doolittle (17) and Ainsworth (1) have found the watermelon usually resistant to the disease, but Ainsworth reports successful transmission of what is probably this virus to *C. vulgaris* from mosaic vegetable marrow. Occasional diffuse pale yellowish spots developed on the leaves inoculated and systemic symptoms took the form of few to many rather inconspicuous pale greenish-yellow spots, sometimes with a small necrotic centre, on the leaves. Recently, however, the natural occurrence of watermelon mosaic has been described in Florida, U.S.A. (70). Diseased vines are conspicuous because the tips of the runners and a proliferation of shoots from around the crowns protrude stiffly above the general level of the vines giving what has been described as a "petunia-like" appearance. These tips and shoots show a shortening of the internodes, resulting in crowding of the young leaves which appear somewhat stunted and more rolled than normal. Some mottling is evident on the leaves, the mottle being of a diffuse type consisting of irregular yellow areas only slightly lighter in colour than the normal light green of the young leaves. On some plants the young leaves show extreme malformation, being reduced in a few cases to the midrib alone. Older leaves show a more conspicuous mottling in which dark green areas appear, but the mottling is less characteristic

than the severe malformation that occurs on many leaves. In addition to the crowding of the young leaves on the tips there is also a crowding of flowers and flower buds. Some flowers on severely affected plants may show abnormalities of the petals and floral parts such as unequal lengths and a greenish cast to the petals. Necrosis and shedding of blossoms also occurs and fruits are few. When present the melons are distorted and show a pronounced mottling of slightly raised dark green areas.

Micrampelis lobata (Michx.) Greene. Wild cucumber. On the wild cucumber there is usually a marked variegation and deformity in the leaves of mosaic plants. In most cases the symptoms are similar to those on the cucumber with the exception that the lighter coloured portions of the leaf predominate. These are a light yellowish-green with an occasional almost colourless area which is in sharp contrast to the surrounding tissue. The other parts of the leaf are a deep green and are raised above the surrounding surface. As a result the savoyed character is usually pronounced and leaves are deeply wrinkled and curled. There is also considerable dwarfing and deformity in mosaic leaves, the lobes being irregular in size and outline. The fruits are of irregular shape with wart-like protuberances and the outer integument is frequently split (17).

Sicyos angulatus L. Symptoms are similar to the foregoing, the mottling of the young leaves is seldom extreme, but the older leaves tend to yellow and die off rapidly, the dead leaves remaining attached to the stem as in the case of the cucumber.

Bryonia dioica Linn. Common bryony. The common bryony of the hedgerow is frequently affected with a mosaic disease, the causative virus of which is thought to be *Cucumis Virus 1*. Ainsworth and Ogilvie, however, report failure to transmit this virus experimentally to cucumber. The symptoms on bryony consist mainly of a mosaic mottle.

Bryonia alba. White bryony. This plant has been found naturally infected with mosaic and Ainsworth (1) has isolated *Cucumis Virus 1* from such mosaic plants which showed a yellow mottle.

Histopathology. No intracellular inclusions (X-bodies) have been observed in any solanaceous or other plant infected with *Cucumis Virus 1*. This is a point of difference from the superficially similar diseases produced in the same host plants by *Nicotiana Virus 1*, which does induce the formation of X-bodies.

Euphorbiaceæ

Euphorbia splendens. The "crown of thorns" (see Fig. 12, D).

The writer has observed a specimen of this plant with pale yellow rings upon the leaves. These rings are chlorotic and may be concentric. Inoculation to tobacco from this plant produced in the former symptoms indistinguishable from those produced by *Cucumis Virus 1*.

Leguminosæ

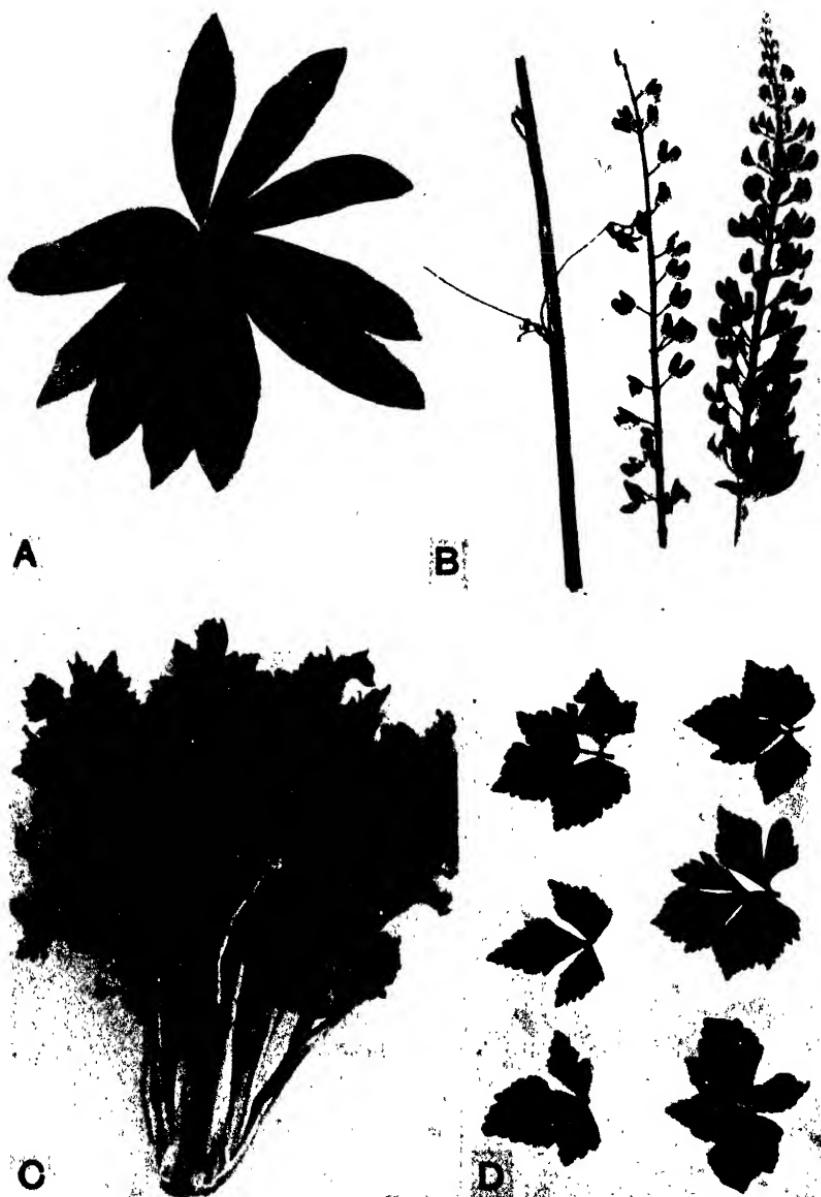
Vigna sinensis. Endl. Cowpea. The leaves of several varieties of cowpea react with local lesions when inoculated with the various strains of this virus. The lesions appear within three to four days after inoculation and consist of circular, dark red necrotic spots. These vary in size, but are usually between 1 and 2 mm. in diameter. There is no further development of the disease as a rule, but Price (49) has isolated a yellow strain of the virus which does become systemic in the cowpea. This strain produces yellow lesions which are not necrotic, but which have a necrotic periphery. Systemic infection takes the form of a green and yellow mottle on the uninoculated leaves accompanied by severe stunting and distortion.

It is possible that the virus also affects the lima bean, producing mosaic mottle and stunting.

Lupinus angustifolius. Lupin. The writer has observed a disease in lupins, caused by *Cucumis Virus 1*, in private gardens in England, while what is apparently the same disease has been described on lupins in Germany (36). The main symptoms of attack are a distortion of the leaves, together with the development of necrotic spots on individual leaflets (see Fig. 11, A) and the appearance of a longitudinal stripe along the stem (see Fig. 11, B).

FIG. 11. *Cucumis Virus 1* (cucumber mosaic virus).

- A. Leaf of infected lupin plant.
- B. Stem and blossom of infected lupin, blossom from healthy plant (right): note the longitudinal lesions on the stem and the individual blossoms widely separated.
- C. Infected celery plant, showing discolouration, necrosis, and shrivelling of the petioles.
- D. Mosaic celery leaflets, showing pronounced mottling that occurs on young leaves, healthy leaflet (right, centre). (C and D, after Doolittle and Wellman, *Phytopath.*)



Infected plants frequently fail to flower; when flowers are present they bear the individual blossoms widely separated.

Umbelliferæ

Apium graveolens L. Celery. A serious disease of celery has been described and studied by Wellman (71) under the name of Southern Celery Mosaic. He considered the causative virus to be a new one, and called it *Celery Virus 1*. In 1931, however, Doolittle (18) suggested that celery mosaic was caused by the virus of cucumber mosaic (*Cucumis Virus 1*), and in a more recent paper Price (51) offers evidence that the viruses causing celery and cucumber mosaic are one and the same. Price shows that infection of zinnia plants with the virus of celery mosaic induces in them a specific immunity from a yellow strain of the cucumber mosaic virus. Further, the respective symptoms produced by the two type viruses in *Zea mays* L., *Commelina communis* L. and *Vigna sinensis* (L.) Endl. are identical. The virus causing "Southern Celery Mosaic" is therefore accepted as a strain of *Cucumis Virus 1*.

On celery the early symptoms of mosaic consist of pronounced outward and downward curling of the younger petioles which gives the heart of the plant a characteristic open, flattened appearance. The leaflets show a greenish-yellow colour along the veins which later develops into whitish areas of irregular outline. The intervening green areas in the lamina remain green, and this colour intensifies with age. In mature leaflets the green areas are somewhat thicker than the yellow portions. The leaflets are therefore slightly savoyed and crinkled. There is no filiformity or other malformation of the leaflets. Mosaic celery plants appear stunted since the leaves tend to lose their upright habit, but the length of the petioles is not greatly shortened, although their width and thickness are much diminished. As the disease progresses, buff-coloured sunken and translucent spots of irregular size and shape frequently develop on the petioles of mosaic-affected plants. The vascular system of petioles thus affected is often discoloured, and in several cases the petioles become brown and badly shrivelled (20) (see Figs. 11, C, 11, D.)

A mosaic disease of celery which is probably due to this virus has recently been described in Italy by Gigante (24), who states that the insect vector in this case is the aphid, *Cavariella pastinacæ*.

Daucus carota, var. *sativa*. The carrot. The carrot is susceptible to infections both by the agency of the aphid and by

sap-inoculation. The symptoms consist of a mottling accompanied by yellowing and some necrosis.

Apocynaceæ

Vinca minor. Periwinkle. The writer has observed this plant in private gardens in England to be infected with *Cucumis Virus 1*, and Wellman (74) records it as a host plant of the virus in the U.S.A. In Florida, where the periwinkle is treated as a perennial, this plant often manages to exist for years with fairly severe leaf symptoms. Infected plants show a pronounced mottling of the leaves with slight stunting. The first symptom of systemic infection is a rather characteristic streaky mottle which later increases in severity. The disease results in downward curvature of the leaves and shortened internodes. The flowers, when produced, are small but not noticeably deformed : in the case of blue flowers a white "break" is present.

Asclepiadaceæ

Asclepias syriaca. Milkweed. Mosaic plants are easily recognised by their dwarfed growth and mottled and distorted leaves. Infected plants rarely reach a height of more than 2 feet compared to 3 or 4 feet attained by normal plants. The leaves are mottled with irregular patches of greenish-yellow and rather distorted. They are smaller than those of healthy plants and are usually more lanceolate than the normal oblong leaves. In some instances the diseased leaves are of abnormal length and taper rapidly from a broad base to an almost filiform tip. Commonly half the leaf blade develops to normal size while the other half is only a few millimetres wide for all or part of its length. This effect of the disease produces abnormalities which are readily recognisable. The leaves of mosaic plants also show a tendency to curl upwards at the margins, producing a cup-like effect in extreme cases (19).

Compositæ

Callistephus chinensis. China aster. A strain of *Cucumis Virus 1* affects asters in England and produces symptoms slightly suggestive of aster yellows (*Callistephus Virus 1*). The leaves of affected plants are mottled and chlorotic and may be entirely yellow. The flower heads are small and malformed and more numerous than is usual in a healthy plant.

Calendula sp. Affected plants are mottled and distorted and

the leaves frequently show necrotic areas. The flowers are small and are sometimes misshapen.

Zinnia elegans Jacq. On inoculation to zinnia, clearing of the veins develops after six to eight days, while the systemic symptoms of mottling appear on the tenth or twelfth day. The type of mottling and distortion produced is very similar to that on cucumber and other plants infected with this virus (50) and may be very severe. The colours of diseased zinnia flowers are abnormal with tan and dirty grey markings on the distorted, irregular and stiff petals. The flower stalk is shortened and the numbers of flowers produced are much less than normal (74).

Primulaceæ

Primula obconica and *P. sinensis*. Affected primula plants are as a rule extremely stunted, the young leaves being abnormally small and distorted. There is usually some yellow mottling of the leaves and growth is slow compared with that of healthy plants. The colour of the flowers is sometimes affected and flecks or streaks may develop on the petals.

Polyanthus sp. The virus produces no very characteristic symptoms on *Polyanthus*, but the leaves are chlorotic and the plant is stunted with poor growth.

Campanulaceæ

Lobelia cardinalis. Affected plants of this species (vars. Purple King and Mrs. Hubert) show a pronounced mottling of the leaves which takes the form of a spotty or patchy mosaic of dark green with paler spots. The youngest leaves are distorted and twisted, the older leaves are also somewhat malformed and rather brittle. The colour of the flowers does not appear to be affected.

Solanaceæ

Nicotiana tabacum, var. White Burley. Pale green circular spots may appear on the inoculated leaves two or three days after inoculation, but no necrotic lesions are produced. Systemic infection first shows as a slight clearing of the veins, and this is followed by a mild general mottle. There is sometimes distortion (narrowing) of the leaves (1). On the whole the symptoms produced on tobacco by the type virus are mild, though some of the strains of this virus produce more severe effects.

N. glutinosa. On this plant some necrosis may be produced,

the mottling is sometimes severe and there are occasionally dark green blisters on the leaves (see Fig. 10, B). The whole plant is stunted.

Solanum nigrum. Ainsworth (1) states that he has failed to infect this plant experimentally. The writer has isolated the virus from an unidentified solanaceous weed which much resembled *S. nigrum* except for a slightly increased hairiness of the leaves.

Lycopersicum esculentum. Tomato. *Cucumis Virus 1* produces in tomato the disease known as "fern leaf," where the lamina of the leaf is reduced or absent. Such symptoms, however, are not restricted to infections with this virus alone, but are also caused by *Nicotiana Virus 1* and certain of its strains.

The first characteristic symptom of the disease appears about ten days after inoculation and consists of a spindling appearance of the young leaves in the terminal bud. These leaves twist round in a corkscrew fashion; the young leaves which in a normal plant start to unfold at an early stage, remain folded, curve downwards or curl up in spirals. Chlorosis of the older leaves, especially along the veins, is also an early symptom. About three weeks after inoculation, the spindling leaves have unfolded, and typical distortions are formed which result in filiform leaves. These leaves, of which two or three originate from the terminal bud, are characterised by marked reduction of the lamina, often to such an extent that only the mid-rib is left. Besides the filiform leaves developed in the terminal bud, several others may be formed in the leaf axils. A marked rolling and folding of the leaves as well as a characteristic mottling often accompany these early symptoms. About five weeks after infection, when the filiform leaves are well developed, another type of symptom becomes apparent. This symptom is conspicuous by the excessive number of lateral leaflets produced (42). The foregoing description is applicable to tomatoes affected with the American strains of the virus. In England the symptoms on tomato are usually less pronounced, and a very mild mottle without distortion or with only slight narrowing of the leaves are the usual symptoms. Occasionally, however, the true "fern-leaf" disease is produced. Mogendorff (42) states that he could produce the fern-leaf type of symptom at will by using the aphid *Myzus persicae* as the infecting agency and he also showed that air temperature affects the type of symptom, the minimum, optimum and maximum air temperatures for the expression of fern-leaf being 15° C., 18° to 22° C., and 25° C. respectively.

Capsicum annuum. Pepper. The younger leaves of the infected plant curl downward along the mid-rib and the basal portion of the leaf is frequently a lighter green than that at the tip. Within a short time, however, such leaves develop the mottled appearance characteristic of the disease. In pepper plants long infected, the leaves are considerably smaller than those of healthy plants of the same age. The stem internodes are shortened considerably and the length of the petioles is somewhat reduced. Infected plants have a more compact habit of growth than normal plants and the leaves are often abnormally narrow and drawn out in filiform fashion at the tip. The character of growth often produces an almost rosette-like character in plants which are infected when young. The foliage becomes a yellowish-green and the leaves appear to be firmer in texture than those of healthy plants. Occasionally the fruits of infected pepper plants show symptoms somewhat similar to those found in the fruits of the cucumber. The greater part of such fruits retain their normal green colour, but the surface is broken with dark green areas which are raised above the surface in warty swellings similar to those found on affected cucumbers (19).

Petunia sp., var. Rosy Morn. Affected plants have mottled leaves which may develop some necrotic areas. The stem internodes become much shortened and under certain conditions distinct necrotic streak symptoms appear on them. The leaf blade is much distorted and blossoms are few or absent. The virus can be transmitted to this species by sap-inoculation and by the aphis (*A. gossypii*), and in each case primary lesions develop on the inoculated leaves. These start as small whitish spots which enlarge rapidly, becoming slightly yellow and finally water-soaked and necrotic. Symptoms of systemic infection appear on the leaves several days after the development of the primary lesions and consist of clearing of the veins and later of severe mottling and stunting. Necrosis extending to the stem may sometimes

FIG. 12.

- A. Leaf of chrysanthemum affected with *Lycopersicum Virus 3* (tomato spotted with virus).
- B. Leaf of *Commelina nudiflora* affected with *Cucumis Virus 1* (cucumber mosaic virus).
- C. Leaf of pineapple affected with *Cucumis Virus 1*.
- D. Leaf of *Euphorbia splendens* affected with *Cucumis Virus 1*.

(A, after Ainsworth ; B, after Doolittle and Wellman ; C, after Carter.)



develop. The flowers may also show effects of the virus infection, in some cases they may be only partially developed, being unable to open completely. Alternatively they may open normally, but are mottled with streaks and irregular concentric rings (74).

Scrophulariaceæ

Pentstemon. The writer has observed this garden plant in England naturally infected with *Cucumis Virus 1*. There is a fairly marked mosaic mottle on the leaves which tend to curl downwards at the apex. Infected plants are much stunted in comparison with normal plants.

Pedaliaceæ

Martynia louisiana. Infected *Martynia* plants develop a curling and mottling of the younger leaves which is similar in appearance to the disease caused by this virus in cucumber plants except that the green areas are very large and few in number, the yellowed portion including the greater part of the leaf. Occasionally the virus becomes localised in a portion only of the plant which alone shows the disease symptoms. Infected plants are much dwarfed and the older leaves tend to wilt and die. In most cases the fruits are much dwarfed and show no mottling but rarely, plants occur whose fruits are slightly mottled with yellow and show wart-like protuberances somewhat similar to those found on affected cucumber fruits (19).

Monocotyledons. Commelinaceæ

Commelina nudiflora L. Creeping dayflower, Wild Wandering Jew. The leaves of affected plants are distinctly mottled with areas of greenish-yellow present either as rather large, irregular longitudinal patches or as roughly circular spots from 2 to 4 mm. in diameter. These yellow areas often extend across the veins and the mottling is not confined to the sharply defined longitudinal striping that characterises most mosaic diseases on monocotyledonous hosts (20) (see Fig. 12, B).

Liliaceæ

Until just recently lily mosaic has been considered to be due to the action of a distinct virus, and in the manuscript of this book it was originally classified as *Lilium Virus 1*. Price, however, has published a paper (52) in which he shows that zinnias infected



FIG. 18. *Cucumis Virus 1* (cucumber and lily mosaic virus).
Plant of *Lilium speciosum*, showing mosaic mottling. (After Cotton.)

with the virus from mosaic lilies are immune from infection with *Cucumis Virus 1*. The name *Lilium Virus 1* is therefore dropped and lily mosaic virus is included as a strain of *Cucumis Virus 1*.

Lilium longiflorum and *Lilium* spp. Lilies. Mosaic is found in all the commercial varieties of *L. longiflorum* which in the British Isles is nearly always grown under glass. In mild cases the mottling consists of faint, irregular, pale green streaks on the dark green leaves which are of normal size and form. In more severe attacks, the leaves, in addition to being mottled, are distorted, and, in later stages, dead spots appear in the pale areas. The effect on the flower is seen in spotting and crinkling and in the perianth segments cohering at the tips so that the buds do not open. Owing to the improvement in quality of imported bulbs, severe cases of infection are not now so common ; where they occur there are streaks of dead tissue in the leaf, dwarfing of growth rather resembling the disease caused by *Lilium Virus 1* (lily rosette), and serious puckering and distortion of the flowers. With high temperatures or when the plants are being hard forced, the mottling is less marked, or may be completely suppressed, and the stunting effect may be reduced, but there is no improvement with regard to the flowers. The bulbs of mosaic-diseased plants are smaller than normal and somewhat flaccid, but such relative characters are difficult to define, and variations in size and crispness may also be due to other causes.

The disease is found in *L. speciosum*, in which it is serious, and in *L. auratum*, where it often causes complete failure (see Fig. 13). It is plentiful in *L. candidum* and *L. testaceum*, in both of which it leads to serious deterioration. In certain stocks of *L. tigrinum* the disease is rife, but in this lily, as well as in *L. Humboldtii* and *L. croceum*, the attack is usually not severe and resembles in form and intensity that found in mild attacks in *L. longiflorum*. In *L. auratum* and *L. speciosum* the mosaic symptoms are very marked, and the effect of the disease is more serious. The mottling is much coarser than in *L. longiflorum* and shows itself as elongated yellow streaks and blotches resembling a form of variegation. The obscure "dying off" which is met with in these two species is believed to be due almost entirely to the virus, and the effect on the plant is so serious that normal flowers are seldom produced.

L. candidum shows a wholesale deterioration owing to mosaic infection both in England and the United States. Affected plants show, especially in spring, an indistinct mottling of the leaves and

often have a slight yellow cast. The mottling can be more easily seen if a leaf is detached and held to the light. In summer the symptoms are usually masked.

In America, *L. regale* and *L. harrisii*, the Easter Lily, are also frequently infected (15).

Control. Methods for the control of lily mosaic are largely confined to hygienic measures of cultivation, since no resistant varieties are known at present. For lilies grown under glass, the careful eradication of diseased plants is important together with routine nicotine fumigation to destroy the insect vector. In the open, efforts should be made to keep down aphides as far as possible while all diseased plants should be carefully rogued out. It is advisable not to include *L. candidum* in the general lily collection, since this species is so frequently infected. In vegetative methods of propagation care should be taken that no bulbils or scales used for this purpose are from diseased plants. Propagation by seed is a useful method of raising a stock of virus-free plants, since the virus is not seed-transmitted in the lily. The removal of diseased plants from the nursery beds is important, and eradication should be commenced early and continued at frequent intervals throughout the season (15).

Allium cepa L. Onion. The first visible signs of infection appear as a number of light chlorotic streaks on the young leaves. Older leaves become chlorotic with yellowish markings of rounded and irregular ring shapes. The virus appears not to be sap-inoculable to this plant, but is transmitted by the aphis. Six weeks after the time of infection, diseased plants have become yellowed, stiff, brittle and somewhat stunted (74).

Musaceæ

Musa sapientum. Banana, var. Lady Finger. In experimental infection of medium-size heads or corms, early symptoms are usually first noticed on the third leaf appearing after infection. This leaf changes little in size and general growth habit from the normal, though it is usually chlorotic, slow to unfurl, and drooping, and may be severely marked with yellowish areas interspersed with greener regions on the leaf blade. Succeeding diseased leaves are tightly rolled, and only two leaves are pushed out on the affected plant in the time taken by a healthy plant to produce four fully developed leaves. Diseased leaves are brittle, easily torn and have chlorotically spotted petioles with malformed fibro-vascular bundles, and necrotic spots and streaks often occur on the leaf

lamina and pseudo-stem. There is distinct reduction of petiole and leaf blade and a consequent stunted rosette-like appearance, resembling the symptoms of banana bunchy-top (*Musa Virus 1*). When the plants become diseased the leaf sheaths are considerably reduced in rapidity of growth and flexibility. Partial strangulation of the newest leaves in the centre of the pseudo-stem causes their malformation and occasional splitting of the leaf sheaths (73).

Graminaceæ

Zea mays. Maize. In experimental infection of maize seedlings the incubation period of the disease varies from three to twenty days. The most rapid and severe infection occurs at temperatures between 70° and 90° F. Primary lesions about 1 mm. in diameter appear in three days around the feeding punctures of viruliferous aphides. About two days later the lesions spread downward along the veins of the seedling leaf. Faint systemic symptoms occur about eight days after inoculation and four days later systemic symptoms are distinct. The early symptoms consist of numerous light-coloured, intermittent, elliptical spots of various lengths and widths whose long axes are parallel with the veins of the leaves. As infection becomes severe, the plants are badly stunted and are very similar to plants infected with Cuban white stripe (64). They lack, however, the severe rosette symptoms of that disease, and the striping of the leaves is more translucent with distinct demarcation between chlorotic and green areas. There is a tendency to leaf splitting and sometimes crumpling of the leaf tips. Mottling is found on the first systemically infected leaves and buff-coloured and necrotic areas on old severely affected leaves (72).

Strains of Cucumis Virus 1

Cucumis Virus 1A

Synonym. *Cucumber Virus Strain 5*, Price, 1934.

This virus produces symptoms which are very different from those caused by the type virus. It causes necrotic primary lesions in tomato, *Nicotiana glutinosa*, *N. langsdorffii*, Turkish tobacco and spinach and yellow and necrotic primary lesions in cucumber. The first four of these hosts develop a systemic necrosis when infected. The necrosis is usually present as solid spots or zonate rings, but may occasionally follow the veins and produce oak-leaf patterns (49).

Cucumis Virus 1B

Synonyms. *Cucumber Virus Strain 6*, Price, 1934; *Yellow Cucumber Mosaic Virus*, Price.

This strain produces the most brilliant symptoms of any of the strains of this virus. It causes yellow primary lesions in Turkish tobacco, cucumber, spinach, tomato, *Nicotiana glutinosa* and *N. langsdorffii*. Systemic symptoms in all these plants consist of a mottling of brilliant yellow and dark green. Infected leaves of tobacco, *N. glutinosa* and spinach occasionally become almost entirely yellow. More frequently, the yellowing is confined to certain portions of the leaves, the remaining portions being green (49). In *Primula obconica* this strain produces a pronounced yellow and green mottling.

Cucumis Virus 1C

Synonym. *Y Strain Cucumber Mosaic Virus*, Price, 1934.

This strain differs from the type virus and from all other strains by its ability to produce a systemic disease in cowpea (*Vigna sinensis*), var. Black Eye. Yellow primary lesions are produced, and in many cases, but not all, a necrotic ring partially or completely encircles the lesion. Systemic infection takes the form of a severe mosaic consisting of a green and yellow mottle.

Cucumis Virus 1D

Synonyms. *Cucumber Virus 2*, J. Johnson's Classification; "*Bettendorf*" *Mosaic Virus*, Porter, 1930; *Cucumber Mosaic Virus*, Porter, 1931.

Although this virus has been classified by some authors as a distinct entity the writer considers that there is insufficient evidence to justify its grouping as a separate virus and it is therefore dealt with here as a strain of the type virus *Cucumis Virus 1*.

Diseases Caused by Cucumis Virus 1D

This strain was recorded by Porter in 1931 (48), who considered it to be different from the type virus. The differences appear to be mainly in the symptoms produced by the two on hosts of the same strain or variety. The first symptoms caused by this virus occur either on the terminal leaf, or on those receiving the inoculum, in the form of one or more yellow, irregularly-shaped spots. Later these spots increase in number and produce a densely mottled pattern of yellow and green. This condition is similar to the

symptoms produced on cucumbers by *Nicotiana Virus 12*, except that in the latter case the yellow spots are smaller and more circular in outline. No stunting effect is manifest until after the first symptoms appear. The terminal leaves do not droop, but remain rigid throughout the life of the plant. Visible symptoms on the fruit have never been observed. The variety of cucumber Chinese Long which has long slender-necked fruits is extremely resistant to infection with the type virus. In contrast to this is its striking susceptibility to *Strain 1 D*.

Citrullus vulgaris Schrad. The watermelon. Infection on the watermelon with *Strain 1 D* does not occur as readily as on the cucumber, but the fact that the virus is transmissible to watermelon is a point of difference from the type virus. The symptoms on young plants consist of a mottling of the youngest leaves followed by either slight or severe stunting. Older plants show little signs of infection other than a faint mottling of the terminal leaves.

The cucumber virus recorded by Hoggan (28) and designated "cucumber mild mosaic virus" is considered by J. Johnson to be a separate virus which he has called "*Cucumber Virus 5*." The writer considers, however, that for the present it may be regarded as a strain of *Cucumis Virus 1*, and so it is included here in that category.

Host Range of Cucumis Virus 1

Ranunculaceæ

Delphinium sp., *D. consolida* L. Larkspur.
Aquilegia sp. Columbine.

Cruciferæ

Brassica rapa L. Turnip.
Nasturtium officinale. Watercress.

Violaceæ

Viola cornuta. Garden viola.

Polygonaceæ

Fagopyrum esculentum Gaertn. Buckwheat.

Phytolaccaceæ

Phytolacca americana (decandra) L. Common pokeweed or pokeberry.

P. rigida Small. Southern pokeberry.

Chenopodiaceæ

Spinacia oleracea L. Spinach.
Chenopodium murale L. Lamb's quarters.
Beta vulgaris L. Red garden beet.
B. vulgaris cicla. Swiss chard.
Amaranthus retroflexus. Pigweed.

Geraniaceæ

Geranium carolinianum L. Crane's-bill.
Pelargonium hortorum. Bailey. Bedding geranium.

Ficoidaceæ

Tetragonia expansa L. New Zealand spinach.
Mesembryanthemum crystallinum L. Iceplant.

Caryophyllaceæ

Lychnis viscaria L. German catchfly.

Tropæolaceæ

Tropæolum majus L. Nasturtium.

Cucurbitaceæ

Cucumis sativus. Cucumber.
C. melo. Melon, muskmelon.
C. pepo. Vegetable marrow, pumpkin.
C. maxima. Hubbard squash.
C. pepo, var. *condensa*. Cocozelle squash.
Citrullus vulgaris Schrad. Watermelon.
Micrampelis lobata. Greene. Wild cucumber.
Sicyos angulatus L.
Bryonia dioica L. Common bryony.
B. alba. White bryony.
Chayota edulis. Jacq. Chayote.

Euphorbiaceæ

Euphorbia splendens. "Crown of thorns."

Bromeliaceæ

Ananas cosmostus. Pineapple.

Leguminosæ

Vicia faba L. Broad bean.
Vigna sinensis Endl. Cowpea.
Lupinus angustifolius. Lupin.

Umbelliferæ

Daucus carota L. Carrot.
Apium graveolens L. Celery.
Anethum graveolens L. Dill.
Fœniculum vulgare Hill. Fennel.
Petroselinum hortense. Hoffm. Parsley.
Pastinaca sativa L. Parsnip.

Apocynaceæ

Vinca rosea L. Madagascar periwinkle.
V. minor. Periwinkle.

Asclepiadaceæ

Asclepias syriaca. Milkweed.

Convolvulaceæ

Convolvulus sp. Bindweed.
Ipomoea purpurea Lam. Morning-glory.
I. batatas. Sweet potato.

Polemoniaceæ

Phlox drummondii Hook. Phlox.
Gilia capitata. Dougl. Gilia.

Boraginaceæ

Cynoglossum amabile Stapf. and Drum. Chinese Forget-me-not.

Hydrophyllaceæ

Phacelia whitlavia Gray. California bluebell.
P. tanacetifolia Benth. Phacelia.

Compositæ

Callistephus chinensis. China aster.
Calendula sp. Marigold.
Zinnia elegans. Jacq.
Ambrosia elatior L. Ragweed.
Tagetes patula L. French marigold.
T. erecta L. Aztec marigold.
Emilia sagittata Vahl. Flora's paintbrush.

Primulaceæ*Primula obconica.**P. sinensis.**Polyanthus* sp.**Campanulaceæ***Lobelia cardinalis.***Solanaceæ***Nicotiana tabacum.* Tobacco.*N. sanderæ.* Sander.*N. glutinosa* L.*N. sylvestris* Spegaz. and Comes.*Lycopersicum esculentum.* Tomato.*Atropa belladonna* L.*Solanum nigrum.* Black nightshade.*S. aviculare* Forst.*S. melongena* L. Eggplant.*Physalis alkekengi* L. Strawberry ground cherry.*P. pubescens* L. Common ground cherry.*P. peruviana* L.*P. angulata* L.*P. lagascae* R. and S.*Capsicum annuum* L. Tabasco pepper.*C. frutescens* L. Sweet pepper.*Datura meteloides* DC.*D. Stramonium* L. Jimson weed.*Petunia hybrida* Vilm. Petunia.**Scrophulariaceæ***Antirrhinum majus* L. Snapdragon.*Pentstemon.***Pedaliaceæ***Martynia louisiana.***Dipsacaceæ***Scabiosa atropurpurea* L. Sweet scabious.

Monocotyledons. Commelinaceæ

Commelina nudiflora L. Wild wandering Jew.

C. communis L. Creeping day-flower.

Zebrina pendula Schnizl. Wandering Jew.

Tradescantia sp. Spiderwort.

Liliaceæ

Lilium longiflorum Thunb. Easter lily.

L. auratum Lindl. Golden lily.

Allium cepa L. Onion.

Musaceæ

Musa cavendishii Lamb. Cavendish banana.

M. sapientum L. Lady Finger banana.

Graminaceæ

Euchlaena mexicana Schrad. Teosinte.

Zea mays L. Maize, sweet corn.

Holcus sorghum L. Sweet sorghum.

Triticum aestivum L. Wheat.

Secale cereale L. Rye.

Geographical Distribution. The virus is widespread in the United States of America and Canada. It is common in many parts of Europe and has been recorded from Holland, Germany, France and England. In the last-named country it is frequently found infecting ornamental plants in gardens and glasshouses.

Control. Much can be done in the control of this virus disease by strict attention to plant hygiene. Careful and regular fumigation of the glasshouses to keep down aphid vectors is of the first importance, and a good nicotine fumigant is the best for this. When the plants are small, any that show a mottling of the leaves should be removed and burned. When the vines are larger, however, and intertwined, diseased plants should be cut off at the roots and left in position to dry out. If they are pulled away, the contact of the diseased leaves and stems with neighbouring healthy plants in the process of pulling will spread the infection. After handling mosaic plants, the hands should be washed with soap and water before healthy plants are touched, and pruning knives should be sterilised in alcohol or thoroughly washed after cutting diseased plants. The eradication or removal to a distance of alternative host plants of the virus is important. In the United

States the following wild plants can act as sources of infection, the common wild cucumber (*Micrampelis lobata*), the milkweed (*Asclepias syriaca*), the pokeweed (*Phytolacca decandra*) and the wild ground cherry (*Physalis* sp.). In Florida, where this virus causes a serious disease in celery, it is important to remove susceptible weeds, such as *Commelina nudiflora* and *Phytolacca americana* L. (74A). In the British Isles and in Europe generally, solanaceous weeds, such as certain of the nightshades, are susceptible to infection, while the common bryony (*Bryonia dionica*) is also a possible source of disease. In addition it should be remembered that many ornamental flowering plants are susceptible to infection, among these may be mentioned *Primulas*, *Polyanthus*, *Delphinium*, *Asters* and *Lobelia cardinalis*. When practicable, therefore, these plants should not be grown in close proximity to cucumbers, vegetable marrows or other members of the same family. Finally, the use of clean seed, i.e., seed from virus-free plants is recommended.

CUCUMIS VIRUS 2. Bewley

Synonyms. *Cucumber Mild or Ordinary Mosaic Virus*, Bewley, 1923; *Cucumber Green Mottle Mosaic Virus*, Ainsworth, 1935; *Cucumber Virus 3*, J. Johnson's classification.

The Virus

Resistance to Alcohol. The virus is not inactivated by 50 per cent. alcohol in one hour.

Serological Reactions. *Cucumis Viruses 2 and 2 A* are very similar to one another antigenically, and they are also serologically related to *Nicotiana Virus 1*. They differ antigenically, however, much more from *Nicotiana Virus 1* than from one another, and much more than the recognised strains of *Nicotiana Virus 1* differ from each other (3).

Thermal Death-point. The virus survives 80° C. for ten minutes, but is inactivated by 90° C. for ten minutes.

Resistance to Ageing *in vitro*. The virus retains infectivity for one year or longer.

Filterability. The virus can be filtered unchanged through Pasteur-Chamberland candles L₁ to L₇, and through a membrane of 150 μ A.P.D.

Crystallization. From cucumber plants infected with *Cucumis Viruses 2 and 2 A*, Bawden and Pirie (8) have isolated nucleoproteins with a similar chemical composition and similar in many



A



B



C



D

of their properties to those obtained from solanaceous plants infected with *Nicotiana Virus 1*. These liquid-crystalline preparations can infect cucumber plants in dilutions of one in a thousand million.

Transmission. The virus is quite infectious and is readily transmissible by sap-inoculation. The insect vector is not known.

Differential Hosts

Unlike *Cucumis Virus 1*, this virus is apparently not transmissible to solanaceous plants nor to vegetable marrow (*Cucurbita peto*) and *Bryonia dioica*. The host range appears to be confined to the Cucurbitaceæ.

Diseases Caused by *Cucumis Virus 2*

Cucurbitaceæ

Cucumis sativus. Cucumber, var. Butcher's Disease Resister.

The cucumber plant is readily infected by sap-inoculation. Symptoms appear seven to fourteen days after infection as a slight clearing of the veins and crumpling of the young leaves, followed by a light green-dark green mottle, together with blistering and distortion of the leaves and stunting of the plant. The symptoms first appear on the younger leaves, and leaves fully developed at the time of infection show no symptoms. The mottle is independent of the season, but leaf distortion is more severe when the plant is growing slowly in winter. Occasional yellow flecks occur on leaves showing the green mottle and may, on fully developed leaves, be a prominent symptom. The fruit is usually unmarked, though it may be slightly mottled (1).

Cucumis melo. Melon. The symptoms on infected melon plants consist of a dark and light green mottle of varying intensity, leaf distortion and stunting of the plant. Similar symptoms are produced on *C. anguria* (gherkin) and *C. maderaspatanus*.

Citrullus vulgaris. Watermelon (varieties Florida Favourite and Dixie). Seedlings of this species are easily infected,

FIG. 14. *Cucumis Virus 2 A* (yellow cucumber mosaic virus).

- A. Fruit from infected cucumber plant, showing the markings caused by this virus.
- B and C. Leaves from infected cucumber plants, showing two different types of mottling.
- D. Leaf from infected watermelon. (After Ainsworth.)

symptoms are a dark green-light green mottling and slight stunting.

Strain of Cucumis Virus 2

Cucumis Virus 2 A

Synonyms. *Cucumber Aucuba Mosaic Virus*, Bewley, 1923; *Cucumber Yellow Mosaic Virus*, Ainsworth, 1935; *Cucumber Virus 4*, J. Johnson's classification.

The Virus. This strain seems to bear the same relationship to *Cucumis Virus 2* as "aucuba" mosaic virus of the tomato bears to *Nicotiana Virus 1*.

Diseases Caused by Cucumis Virus 2 A

Cucurbitaceæ

Cucumis sativus. Cucumber, var. Butcher's Disease Resister.

The incubation period in the plant is nine to sixteen days, slightly longer than that of the type virus. The first symptoms, a slight clearing of the veins and temporary crumpling of the apical leaves, are followed by a bright yellow mottle, but little or no distortion of the leaves, and the plant is slightly stunted. The mottle either takes the form of few, to very many, dendritic or star-like spots which may almost cover the entire leaf or gives a vein-banding effect. The vein-banding may be well defined or appear as a filigree of fine lines which follow the smaller veins. The colour of the mottle, which is always well defined, varies from a pale yellow or greenish-cream to nearly white on older leaves. Under less favourable growing conditions the bright yellow mottle is replaced by a rather inconspicuous yellowish-green mottle and slight distortion, the symptom picture is then very similar to that given by a plant infected with the type virus. The fruit is marked by yellow or silver-coloured spots or streaks which may be intensified at high temperatures (see Fig. 14).

On other Cucurbitaceæ the symptoms are similar to those induced by the type virus except that the mottling is yellower.

Geographical Distribution. *Cucumis Viruses 2 and 2A* appear to have been recorded only from England (1, 6).

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CHAPTER II

Gossypium Virus 1 ; Manihot Viruses 1-2 ; Ribes Virus 1 ; Fragaria Viruses 1-4 ; Rubus Viruses 1, 2, 3, 3A, 4, 5 ; Holodiscus Virus 1 ; Prunus Viruses 1, 1A, 2-6 ; Pyrus Viruses 1-2 ; Rosa Viruses 1-4 ; Phaseolus Viruses 1-3 ; Soja Virus 1 ; Pisum Viruses 1, 2, 2A-2C ; Trifolium Virus 1 ; Medicago Viruses 1-4 ; Robinia Virus 1 ; Arachis Virus 1.

GOSSYPIUM VIRUS 1. Farquharson

Synonyms. *Cotton Leaf-crinkle Virus*, Kirkpatrick, 1930 ; *Cotton Leaf-curl Virus*, Kirkpatrick, 1931.

The Virus and its Transmission. The virus is not sap-inoculable and nothing is known of its properties. It is not carried in the seed or the soil and the insect vector is the white-fly (*Aleyrodidae*), *Bemisia gossypiperda*, Misra and Lamba (see p. 499). White-flies can pick up the virus from those parts of an infected Sakel cotton plant which show no symptoms and transmit the disease to healthy Sakel. They can only pick up the virus from a newly-infected plant about one day before the appearance of symptoms.

In order to transmit the virus it is not necessary for the white-flies to have fed on "crinkled" plants as adults, provided that they have done so as larvae. White-flies which have picked up the virus remain capable of transmitting it for seven days, and very probably throughout their life. Uninfected adult white-flies can pick up the virus from crinkly Sakel cotton in just over three hours and possibly in a shorter time. Infected white-flies can transmit the disease to healthy Sakel plants in thirty minutes, and the whole process of infection of the white-flies and of the healthy plant can be accomplished in six and a half hours. The virus cannot be transmitted through the egg of the white-fly.

Differential Host

Hibiscus cannabinus. Til. The symptoms of leaf-curl on Til are extremely conspicuous. There are well-marked net-vein enations and the veins are extremely thick and gnarled on the underside. The leaves remain small and are distinctly curled upwards. When a plant has been infected for some weeks the intercostal areas become yellowish, the veins remaining dark

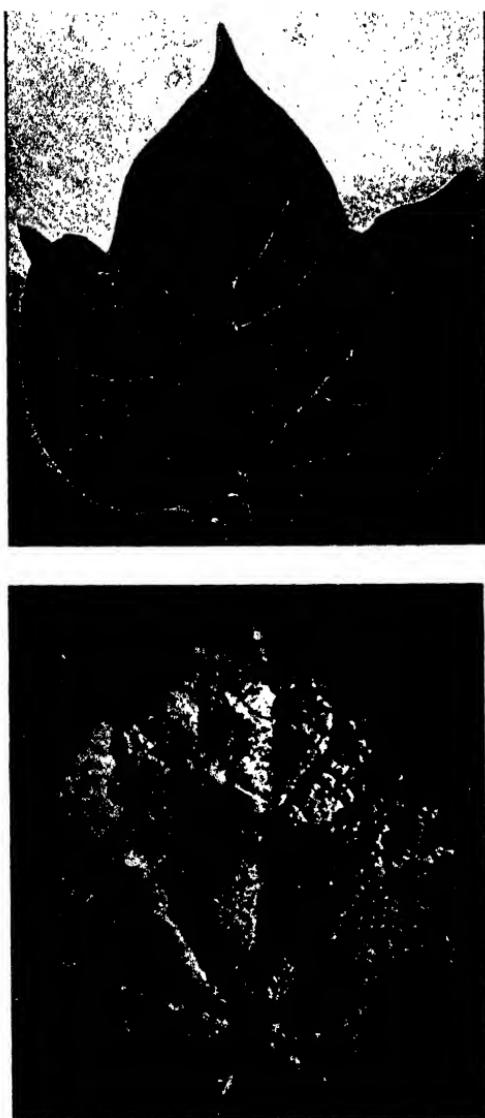


FIG. 15. *Gossypium Virus 1* (cotton leaf curl virus).

(Upper) First symptoms on Sakellaridis cotton : note short lengths of numerous veins darkened.
(Lower) Severe symptoms on Sakellaridis cotton : note cup-shaped foliar outgrowths. (After Kirkpatrick.)

green, giving a mosaic appearance to the leaves. To a certain extent, infected plants of Til combine the features of crinkle, curl and mosaic.

Diseases Caused by Gossypium Virus 1

Malvaceæ

Gossypium peruvianum × *barbadense*. Sakel cotton. Leaf-curl (see Fig. 15). A leaf that is already fully grown never develops symptoms, but a partly grown leaf may develop a thickening of the lower surface of the smaller veins. Such thickening starts at a number of points which gradually tend to join up, until all the veins are affected. Frequently the symptoms first appear on the very young leaves, the older partly grown ones developing normally.

If a diseased leaf is viewed from beneath against the light it will be seen that these thickened veins are darker green than the rest of the leaf and consequently opaque, instead of being translucent, as are the veins of a normal leaf. This character appears to be the best criterion for the presence or absence of leaf-curl, especially when it is necessary to decide if the disease is present in a mild form on a young leaf or an epicalyx, as normally these are somewhat rugose in appearance, but without the pathological thickened and darkened areas on the veins. When an older plant that already has flower buds contracts the disease, it is often the epicalyces of the buds that show the first signs of thickening of the veins.

In some plants at a variable time after the appearance of the first signs of infection, the symptoms become greatly intensified. The new leaves produced are small, exceedingly crinkled, and curled at the edges, either upward or downward. Not infrequently oval cup-like foliar growths are produced on the greatly thickened lower sides of the primary veins. The primary stem of the plant often tends to grow taller than normal, the internodes being elongated and irregularly curved; but sometimes the whole plant is stunted in growth. All parts of a badly affected plant are very brittle and readily broken (49).

The symptoms of leaf-curl differ slightly on other varieties of cotton such as those grown in Nigeria, *G. peruvianum* and *G. vitifolium*. In these the most important characteristic is a strong upward curl of the leaf margin. Other points in which the manifestation of the disease on Nigerian cottons differs from that on Sakel are the absence in the former of any twisting of the

petioles and the rarity of net-vein enations on the bracteoles, a practically universal feature of the disease in Sakel, even if a plant is otherwise only mildly affected. Also the leaves of affected Sakel cotton are never "savoyed" light and dark green. Cotton plants are susceptible to infection at all stages of growth.

Effect on Yield. The disease causes a highly significant reduction in the number of green and open bolls and the unit weight of the green bolls on the healthy plants is significantly greater than that of the green bolls of the infected plants (3).

Gossypium hirsutum. Watts Long Staple cotton. On this variety of cotton the virus produces a mosaic disease rather than leaf-curl. The first sign of infection is a clearing of the veins, the larger of which appear broader than normal owing to a slight chlorosis which tends to be concentrated close to their sides. After a few days the chlorotic areas increase in size, and though often more numerous near the sides of the main veins, thus giving some appearance of symmetry in their arrangement, they also occur scattered at random over the whole leaf surface, so that the leaf becomes more or less uniformly mottled. The pale spots always seem to originate from a veinlet and are not found isolated in the intercostal areas. They are more readily visible on the upper surface of the leaf than on the lower, and they vary in colour from a slightly paler green than the normal leaf to a light yellow or almost white. The chlorotic spots frequently coalesce to form large pale patches which may cover as much as half or even more of the leaf area. The upper surface of the leaves sometimes presents a blistered appearance and their margins are often puckered. The leaves may become asymmetrical owing to a greater concentration of chlorotic spots on one side or the other. There is some tendency towards a bunchy growth, due to the internodes being shortened in a severely affected plant.

Hibiscus esculentus. Bamia. The symptoms of the disease on Bamia are very similar to those on Sakel cotton, the net-vein enations are very conspicuous, but large cup-shaped outgrowths, such as are common on a badly infected Sakel plant, have not been noticed. There is little abnormal curling of the leaves unless a plant is very severely affected, when the leaves are small and thick and curled downwards more often than upwards. It is very difficult to transfer the virus back again to Sakel from Bamia.

Hibiscus sabdariffa. Karkade. The symptoms on this plant are similar to those on *H. cannabinus*, Til.

Althaea rosea. Hollyhock. The symptoms of the disease on

hollyhock are similar to those on Bamia and severely affected plants may have a marked "bunchy top" appearance.

Geographical Distribution. The virus seems to be confined to the Sudan and to Nigeria. The leaf-curl diseases described in Fiji and Italian Somaliland appear to be due to other causes.

Control. Apart from the selection of resistant strains of cotton (see below), the best methods for the control of leaf-curl in the Sudan lie in the destruction of the white-fly, a task which has yet to be achieved, and the removal of the initial sources of infection early in the season. It is believed that the virus is carried over from one season to the next mainly in the ratoon cotton plants which survive the "dead" season and produce new growth, which is almost always diseased, when the land which has been under cotton is irrigated in August for the sowing of Lubia and Dura. It therefore seems probable that if it is possible to eradicate ratoon cotton from August onwards, the reservoir of infection will be abolished. With this end in view a new rotation of crops has been suggested, i.e., (1) cotton, (2) fallow, (3) fallow. The necessary Dura will be sown year after year on land specially set apart. The land from which the cotton has been cut out will, therefore, not be irrigated and "ratoon" plants should not come up unless the rainfall is exceptionally heavy (50). The actual removal of all ratoon plants where this is practicable is also to be strongly recommended.

Resistant Varieties of Cotton. A good deal of success has already been achieved in producing varieties of cotton resistant to the leaf-curl disease in the Sudan. Two types of Sakel cotton, named X1530 and X1730, have proved extremely resistant to the virus under field conditions while still retaining the qualities of vigour and fruitfulness (5).

MANIHOT VIRUS 1. Dammer

Synonyms. *Cassava Mosaic Virus*, *Cassava "Krauselkrankheit" Virus*.

The Virus and its Transmission. There seems to be no information at present on the physical and other properties of this virus. According to some authorities the virus is transmissible by mechanical means. Lefevre (57) states that juice from diseased plants injected into the leaf parenchyma and sub-epidermal tissues of young shoots of healthy plants produced the disease. Another method of mechanical transfer of the virus is by replacing

a narrow strip of tissues down to the wood of the stem of a healthy plant by tissues from the internode of a mosaic-diseased plant.

The insect vectors are one or more species of white-fly (*Aleyrodidae*) ; in Southern Nigeria the species is *Bemisia nigeriensis* Corb (30). In the Belgian Congo the vector is said to be a new variety of a species, *Bemisia gossypiperda*, var. *mosaiceveta* (57). In Amani also a species of white-fly is the vector of cassava mosaic.

Disease Caused by Manihot Virus 1

Euphorbiaceæ

Manihot utilissima. Cassava. *Cassava mosaic*. Affected plants are stunted while the diseased leaves are small and with lobes which are markedly asymmetrical in relation to the nervation. The healthy part of the leaf blade develops more rapidly than the diseased portion, and finally outgrows it, producing a curvature of the veins. At the stalk end where the symptoms first appear, characteristic yellow spots become visible on the upper surface of the leaf which also shows wart-like lesions (24). In Java, the basal leaves of affected plants appear normal, but those immediately above show a pale green to yellowish-green discolouration, sometimes affecting only half a segment or only one or two segments of a leaf. In the upper leaves more especially, the diseased parts are much reduced in size, the leaf blade consisting merely of a narrow strip along the main vein. Affected leaves often show marked curling and distortion, and the internodes are much shortened, giving the plants a stunted appearance. Very often most of the eyes on a stem grow out so that the stem bears an unusual number of lateral branches (62).

Host Range. Other species of *Manihot* seem to be susceptible to the virus, notably *M. aipi*, *M. dulcis* and possibly *M. glaziovii*.

Geographical Distribution. The disease was first described in East Africa in 1885 by Dammer. It is now widespread over East and West Africa, especially in the Gold Coast. It is also prevalent in the Belgian Congo, French Cameroons, Liberia, Java and Madagascar.

Control. There seems to be some possibility of breeding resistant varieties of cassava, since there already exists a certain amount of resistance in some varieties. It is said that the bitter types of cassava are more susceptible than the sweet ones. In Sierra Leone two varieties, Mayugbe and Two Cent, seem to be resistant, while in Java seedlings derived from the Brazilian

"Mangi" strain appear to be particularly susceptible. On the Gold Coast, two varieties, Calabar II and Bankye Saresco, are thought to be useful as breeding stocks.

In Amani recent studies have emphasised the complexity of the problem of the control of cassava mosaic. The prospect of breeding from local cassava varieties new varieties resistant to mosaic is unfavourable. In the hope of introducing new factors for resistance, cassava varieties from West Africa, the West Indies and Java have been introduced. A suggestion made by the Director of the Imperial Mycological Institute is that resistance should be sought in interspecific crosses in the genus *Manihot* (82).

Roguing of diseased plants should be carried out where this is practicable. Where the diseased plants are very numerous, crop rotation should be combined with the careful weeding-out of all plants that grow up from the small pieces of root left in the ground, until the land is completely freed from the crop, when cassava may be grown again.

Strains of Manihot Virus 1

Storey (82) has observed that the cassava mosaic virus in Amani exists in what appear to be strains and that these strains fall into two clearly marked groups, those that produce a severe yellow mosaic, with extreme distortion of the leaves and general stunting, and a second group that produces only a mild green mosaic, with probably little general ill effects. Storey states, however, that it is merely an assumption, unsupported by experimental evidence at present, that the mild strains are in any way related to the severe strains of the mosaic virus.

MANIHOT VIRUS 2. Storey

Synonym. *Cassava Stem Lesion Virus*, Storey, 1936.

The Virus and the Disease Caused by it. This suspected virus disease of cassava has been briefly reported by Storey (82), and there is little information as yet concerning it. The disease has been transmitted by grafting, but the insect vector, if any, is not known.

The chief characteristics of the disease are the production of dark brown stripes on the otherwise green stems, and a yellow mottling of the leaves, which develops only during the later stages of the individual leaf's life. The stem-lesions remain as sunken areas when the stem barks over. If badly diseased, the stem

becomes brittle and readily breaks off at ground level. The leaf mottling is of a different character from that in mosaic (*Manihot Virus 1*) ; the two diseases are readily distinguishable, furthermore, because whereas the mosaic pattern is present on young leaves as they unfold, the young leaves in the disease caused by *Manihot Virus 2* are normal and only develop the mottle on ageing.

The disease has so far only been recorded from Amani, East Africa.

RIBES VIRUS 1. Amos and Hatton

Synonym. *Reversion Disease* of Black Currants.

The Virus and its Transmission. Experiments (2) seem to have shown that the causative agent of this disease is not transmissible by sap-inoculation or by the pruning knife, but can be transmitted by grafting. Furthermore, it seems now fairly certain that the big-bud mite (*Eriophyes ribis*) (see p. 548) is at least one agent in the transmission of the disease. It is also possible that aphides may be additional vectors of the virus. Transmission of reversion through the seed has not been demonstrated.

Disease Caused by Ribes Virus 1

Grossulariaceæ

Ribes nigrum. Black currant. The disease caused in *Ribes nigrum* by this virus is known as "reversion," a condition in which the character of the leaf is changed and the bush appears to have reverted to the wild type. In reality, however, such a bush has not reverted at all, but has become affected with a disease which displays many of the characteristics of a virus infection (see Fig. 16).

The general tendency of the "reverted" leaf is to become relatively narrower and smaller and to "flatten out" at the base, owing to the reduction in depth of the basal sinus or indentation at the base of the leaf (see Fig. 17). Another characteristic feature of the disease is the difference in texture of the leaf surface between normal and reverted leaves. While the normal leaf is provided with a fine network of subsidiary veins and in consequence is relatively smooth-surfaced, the reverted leaf is notably deficient in them, with the result that the surface of the latter is more coarsely rugose.

Many of the lateral buds which should normally produce fruiting trusses only, break into wood growth and produce a crowded



FIG. 16. *Ribes Virus 1* (causing reversion of black currant). Figure showing topmost branch of partially reverted bush typically diseased and barren. (After Amos and Hatton.)

appearance known as "nettlehead." Care should be taken, however, in using this habit of growth as a diagnostic character of reversion, since nettleheaded growths are not infrequently produced on healthy bushes by other causes such as, for example, accidental injury.

As regards the blossoming of reverted bushes it is frequently the case that the flowers are abnormal in that they appear almost transparent, lack the normal pubescence, and have the under side of the sepals of an unusually high colour. These abnormal flowers, however, are not an invariable characteristic of reverted bushes.

Fruits of black currant plants suffering from reversion either do not form at all, the flowers themselves shrivelling up and dropping off, or the fruits may begin to form, in which case development is soon arrested and the half-formed berries shrivel and fall.

Histopathology. As regards the effects of the disease on the tissues, no actual qualitative differences in structure have been observed between normal and reverted plants of *Ribes nigrum*. Those that have been observed are rather quantitative, i.e., differences in degree only, such as amount of gum present and variations in the amount of wood and medullary ray tissue. Briefly, the main differences showing to a greater or lesser degree practically all through the plant are a reduction in the amount of wood, and a consequent increase of medullary ray tissue coupled with a tendency to produce more gum in the revert plant than in the normal (1).

Diagnosis of the Disease

A reliable method of diagnosing reversion in black currants has been evolved by Lees (56). If less than five (submain veins) are present (on each side of the main vein in the central lobe of the leaf) the leaf is reverted, if five or more the leaf is probably normal (see Fig. 17). With regard to the marginal serrations or teeth which do not receive these submain veins and which Lees describes as "uninnervated points," if the margin (on both sides of the central lobe) is finely toothed and if from four to eight of these teeth do not each receive a submain vein, the leaf is probably normal. If, however, this portion of the margins is coarsely toothed, and if less than four of the teeth do not each receive a submain vein, the leaf is reverted to some degree (Leaflet 877, Min. Agric., London).



FIG. 17. *Ribes Virus 1* (causing reversion of black currant).
(Left) Normal leaf of black currant, var. French Black ; note the number of veins and teeth in the terminal lobe.
(Right) Reverted leaf of black currant, var. French Black ; compare the number of veins and teeth in the terminal lobe. (After Amos and Hutton.)

Since the leaves on fruiting growths and short lateral growths are different in character from the fully developed leaves on a strong growing shoot, even in a normal plant, it is necessary, therefore, to diagnose reversion by studying the leafy vegetative shoots which have developed from buds formed in the previous season, and are situated chiefly towards the top or the base of the previous year's wood (1).

Prevention and Control of Reversion. The chief method of prevention lies in the elimination of the sources of infection. This is best achieved by examining the plants in June and July for the leaf characters of reversion. Plantations should be rogued before the crop is picked, the decreased fertility of the reverted bushes giving an additional clue. It has been found that if roguing is carried out regularly at this season and all reverted bushes and branches automatically cut out when diagnosed, the spread within a plantation can be effectively prevented. It is perfectly safe to plant a healthy young bush in the place of a reverted one which has been grubbed up since there is no evidence of soil transmission.

To control attacks of the big-bud mite, bushes should be sprayed with winter strength lime sulphur (s.g. 1·025, approximately 1 gallon concentrate in 12 of water) when the leaves are about an inch across and the blossom trusses are just appearing. The bushes will be covered with a deposit of sulphur which is deadly to mites, at the latest possible moment from the cultural point of view and just when mite migration is in full swing. Some marginal leaf-scorch results, especially on the Goliath group (Victoria, Edina and Monarch); in this group the strength of the spray can be reduced to 1 gallon lime sulphur in 20 of water.

Tar distillate washes and soft soap and nicotine may be used against other possible vectors of the disease (2).

Geographical Distribution. The disease is widespread in the British Isles and probably throughout Europe.

FRAGARIA VIRUS 1. Plakidas

Synonyms. *Strawberry Yellow-edge Virus*, Harris, 1933; *Strawberry Xanthosis Virus*, Plakidas, 1927; *Strawberry Yellows Virus*, Plakidas, 1926; *Strawberry Virus 1*, J. Johnson's classification.

The Virus and its Transmission. The virus causing yellow-edge or "xanthosis" of strawberries is not transmissible by sap-inoculation, but can be spread by grafting. It is not seed-

transmitted. The insect vector in California is the strawberry aphid, *Capitophorus fragefolii* Ckll., and in Europe the same species (see p. 528). This insect is also the vector of what is probably the same disease in New Zealand (17). There is some confusion as to the identity of this aphid, and Hille Ris Lambers (55) considers that the correct name is *Pentatrichopodus potentillae* Wlk. There is no information on the physical and other properties of the virus.

Differential Host

Harris has shown by grafting experiments that the wild strawberry (*Fragaria vesca*) is an extremely sensitive indicator plant for this and the subsequent virus (*Fragaria Virus 2*). Infected plants of *F. vesca* develop the typical symptoms of the yellow-edge disease, they do not show localised necroses (compare *Fragaria Virus 2*).

Diseases Caused by *Fragaria* Virus 1

Rosaceæ

Fragaria vesca. Strawberry. *Yellow-edge*. The disease described by Plakidas in America as "strawberry xanthosis" and that described by Harris in England as "strawberry yellow-edge" are here dealt with as one disease because of the close similarity of the respective symptoms and because grafting experiments to the English indicator variety Royal Sovereign support the assumption that both diseases are identical (87). According to Harris (82) the chief leaf symptoms consist of a chlorosis or yellowing confined to the marginal regions, invariably accompanied by (a) a general dwarfing, (b) an irregular curling of the marginal region, usually in an upward direction, (c) a downward curling of the mid-rib, and (d) a twisting of the whole lamina. A complete leaf has a distorted and asymmetrical appearance due to a combination of the above symptoms on the constituent leaflets. The petiole is abnormally short, stout and lacking in red pigmentation. The intensity of these symptoms varies according to the stage of attack which has been reached (see Fig. 18).

Complete symptoms may appear on individual plants at any time during the season, but in a population of diseased plants the highest proportion showing diagnostic symptoms is generally reached from mid-September onwards. During this period the general appearance of an infected plant is characteristic. This is due to the fact that the marginal chlorosis ("yellow-edge"), lack of red pigment and general dwarfing and curling are limited to the



FIG. 18. Maiden "Royal Sovereign" strawberry plants artificially infected as runners (1) with *Fragaria Virus 1* (yellow-edge virus) and (2) with *Fragaria Virus 2* (crinkle virus). (After Harris.)

youngest leaves. The result of this is the production of an abnormally flat plant consisting of a zone of more or less normal outer leaves enclosing a central zone of dwarf yellow-edged leaves. In cases of mild infection the central leaves and petioles are only slightly dwarfed and curled and only partially deficient in red pigmentation. In advanced cases the central zone consists of a tight rosette of extremely dwarfed, curled and chlorotic leaves entirely lacking in red pigmentation.

Another characteristic of the disease in certain varieties such as Royal Sovereign is the premature reddening or development of autumnal colour as compared with healthy plants. The fruit is not directly affected. These symptoms have been observed in England on the varieties Royal Sovereign, Laxton, Deutsch Evern, Jacunda, Madam Kooi, Flandern, Bedford Champion, King George V., Sir Joseph Paxton, Oberschlesien, Western Queen, Jardine de Leopold and Pillnitz. The variety Stirling Castle showed less leaf-curling and distortion, while the variety Stirlingworth showed more than the foregoing varieties. In California, varieties which show what may be called typical symptoms are the Marshall and Marshall-like strawberry types grown in the central coastal region. Varieties which show a certain degree of resistance to the disease do not exhibit all the typical symptoms (73). The easy recognition of the disease may be obscured by attacks of mite (*Tarsonemus pallidus* Bks.), these induce a chlorosis of young leaves which, however, is spread over the whole leaf and is not, like that of the disease, confined to the leaf margins. In addition chlorosis due to mite attack is associated with a silvery-brown speckly discoloration which is absent in the virus disease. Of the two parent species of the cultivated varieties of strawberry, *Fragaria chiloensis* seems resistant to the virus, or at least shows no symptoms, while *F. virginiana* shows symptoms readily. Cultivated varieties form a complete series between these two extremes. Thus Lefebre very rarely shows symptoms and then very indistinctly, and its susceptibility to deterioration is correspondingly slow.

The development of leaf symptoms on susceptible varieties such as Royal Sovereign is not consistent throughout the season, but is closely correlated (a) with the strain of the virus, i.e., whether "mild" or "severe"; (b) seasonal and accidental fluctuations in temperature and humidity; and (c) soil conditions. In the average season the "seriously" infected plants develop full symptoms early in the growing season, i.e., from early June

onwards, but in the case of "mild" infections the symptoms are generally masked until the autumn months. These remarks apply also to *Fragaria Virus 2* (strawberry crinkle virus) (Harris *in litt.*).

Histopathology. The palisade cells of the chlorotic areas of the leaves are considerably shorter and more compact than those of the healthy plants or of the green areas of the leaves of the diseased plants. A degeneration of the tissue of the pericycle region of the roots is found to be constantly associated with the disease. In this same region cells often occur with partly or completely degenerated nuclei and containing two types of black-staining bodies. These cell-inclusions display no internal structure and are thought to be degeneration products. Neither the degenerating tissue nor the cell inclusions are ever found in roots of virus-free plants (73).

Host Range. The virus is apparently transmissible to most varieties of the cultivated strawberry, though these vary considerably in degree of susceptibility. It is also communicable to the American wood strawberry *Fragaria californica* C. and S., and to the English wild strawberry *F. vesca*. The beach strawberry *F. chilensis* Duch. is highly resistant to infection with high symptom-masking capacity.

Geographical Distribution. The yellow-edge disease of strawberries seems to be widespread, it occurs in the strawberry-growing sections of California, Oregon and Washington. It is common in Europe, particularly England and France, while what appears to be the same disease occurs in the Auckland district of New Zealand (17).

FRAGARIA VIRUS 2. Zeller and Vaughan

Synonym. *Strawberry Crinkle Virus*, Zeller and Vaughan, 1932; *Strawberry Virus 4*, J. Johnson's classification.

The Virus and its Transmission. As will be seen from the discussion on the complex virus diseases of the strawberry, it is not certain that only one virus is concerned in the production of "crinkle," but for the present the disease is treated as if it was caused by a single virus. The virus is not apparently transmissible by sap-inoculation and there is no information on its physical properties. It has been transmitted by grafting (85). The insect vector in America has been shown by Vaughan (85) to be the strawberry aphid, *Capitophorus fragaefolii* Ckll. (see p. 528). In England the insect vector of *Fragaria Virus 2* has not yet been

identified, but it is probably the same aphid as transmits *Fragaria Virus 1* (yellow-edge virus), i.e., *Capitophorus fragaefolii* Ckll.

Disease Caused by *Fragaria* Virus 2

Rosaceæ

Fragaria vesca. Strawberry. Crinkle (see Fig. 18). The two most characteristic symptoms are the crinkling and chlorosis of the leaves. The crinkling is probably due to the uneven distribution of the chlorosis in the early stages of leaf development. The chlorotic areas are at first extremely localised, starting in very small, developing leaves as mere pin-point areas and expanding somewhat as the leaf grows. Frequently small necrotic areas develop in the centre of the chlorotic spots and young leaves bearing this type of stippling may be common on affected plants under unfavourable growing conditions. The chlorosis is localised in spots, and this is the fundamental distinction from "yellow-edge," where the chlorosis is marginal. The veins are frequently cleared. With the uneven distribution of growth there results an uneven margin of the leaflets and the more or less regular indentation becomes a deeper crenation and an unnatural wavy lobing of the margins. At any season of the year affected plants are a lighter shade of green than normal. Under very favourable growing conditions most of the symptoms described above may be lost, but affected plants do not have the uniform greenness and smooth surface exhibited by normal leaves and there is a tendency for the leaves to be downwardly arched or to be cupped upwards at the margins (101).

As regards the crinkle situation in England, Harris (*in litt.*) states that experiments with indicators (differential hosts) show that crinkle is widely distributed in *mild* form throughout apparently healthy strains of Royal Sovereign. "Severe" cases, although hitherto comparatively scarce in south-eastern England, are becoming increasingly prevalent. In the United States of America several seasons may elapse before the disease shows symptoms sufficiently distinct for field identification. Experimentally severe cases of crinkle can, however, be produced by grafting together two plants of different varieties, which are in the "mild" condition of crinkle. Leaf symptoms do not appear consistently throughout the growing season, but are most readily identified in the early part (June) and again in the autumn.

Geographical Distribution. Strawberry crinkle was first observed in 1925 in Oregon, U.S.A. The disease appears to be

widespread in the Pacific Coast States. Infection seems to be particularly common in American strawberries of the Marshall type. Symptoms have also been observed on other American varieties such as Corvallis, Dunlap, Gene, Sear's La Grange, Magoon Missionary and one or two others. In England the symptoms were first recorded in 1934 on the Royal Sovereign variety in the south-western counties by Ogilvie, Swarbrick and Thompson (67), and the virus origin and incidence of the disease in the south-eastern district were later determined by Harris (85).

The Virus Complex

Some recent work by Harris has shown that, in England at any rate, *Fragaria Virus 1* (yellow-edge virus) is almost invariably accompanied by *Fragaria Virus 2* (crinkle virus). Plants showing "severe" crinkle may also be infected with yellow-edge, but such cases are less common than the converse. It is only rarely that the viruses of both diseases act severely in association, but such cases have been induced by artificial infections and have resulted in a condition approximating to that known as "cauliflower."

That the virus situation in strawberries is complex has been clearly shown by recent grafting experiments carried out by Harris, using the common wild strawberry (*Fragaria vesca*) as an indicator plant. This species appears to be extremely sensitive to both viruses, considerably more so than the most susceptible English cultivated variety, Royal Sovereign. The results obtained by the use of this indicator have been confirmed by periodic observation on field experimental plots, and the conclusions reached by Harris may be summarised as follows :

(a) Two distinct types of virus are closely associated with, and widely distributed throughout, the current commercial stocks of Royal Sovereign and other varieties. These types are the yellow-edge and crinkle viruses. The main distinction between these two types is that the crinkle virus produces localised necroses, the yellow-edge virus does not.

(b) Each of these viruses may be present in mild form in carefully field-selected "healthy" plants. "Mild" yellow-edge, however, eventually yields to cumulative infections and can be adequately eliminated from planting stock under suitable conditions. "Mild" crinkle, on the other hand, is still widely distributed in healthy looking stocks.

In experiments carried out during 1936 Harris grafted a number

of " healthy " clonal Royal Sovereign plants to normal plants of *F. vesca* by the " runner-inarch " method. Up to the end of that season all the Royal Sovereign plants remained uniformly vigorous and " healthy," one type of symptom only being detected on a large proportion of the plants, *i.e.*, the minute circular lesions of " incipient crinkle."

On the other hand, the corresponding *F. vesca* plants developed a wide range of symptom types varying in severity from plants which were approximately normal to plants which were drastically stunted. These types can be interpreted as combinations of at least three distinct symptom groups.

- (1) Yellow-edge.
- (2) Crinkle (*a*).
- (3) Crinkle (*b*).

The manifestly complex nature of the crinkle disease explains the clear line of demarcation between what has been described above as " mild " and " severe " stages of crinkle infection. The rapid and hitherto inexplicable passage of one condition to the other is readily explicable on the assumption that the severe phase is the result of the interaction of at least two viruses.

The great variation in the degree of severity in yellow-edge may be in part due to interaction with the crinkle viruses or may be due to the existence of more than one yellow-edge virus.

FRAGARIA VIRUS 3. Zeller

Synonyms. *Strawberry Witch's Broom Virus*, Zeller, 1927; *Strawberry Virus 2*, J. Johnson's classification.

The Virus and its Transmission. The virus is transmitted by the strawberry aphid *Capitophorus fragaefolii* Ckll. (see p. 528). There is no information on other modes of transfer of the virus or on its physical properties.

Disease Caused by Fragaria Virus 3

Rosaceæ

Fragaria vesca. Strawberry. *Witch's Broom Disease* (see Fig. 19). The disease expresses itself differently according to the variety of strawberry affected ; there are two main types of symptom picture, that on the American strawberry Marshall and that on plants of the Ettersburg No. 121 type. In the former variety affected plants have long, unusually erect, stiff, spindly petioles upon which are borne leaflets which are much smaller than those of healthy plants. The leaves are usually light green, with a tendency towards

olive green shades rather than bright green. The mid-veins of the leaflets arch downwards. The lateral pinnate veins do not show this tendency so much, but still enough so that the complete circle of three leaflets has the appearance of curving downward along the entire margin. This is an important point of difference from strawberry yellows, where there is an *upward* cupping of the leaflets. There is a tendency for the stems of individual leaflets to be longer and for the mid-veins to be broader and lighter in colour than in healthy plants. The great number of leaves with long petioles gives the bushy appearance associated with witches' brooms. In the case of Marshall plants, flower stalks are very scarce and, when they do appear, are spindly and unfruitful. The runners are very much shortened and the young plants are thus formed nearer the parent than is normal.

In the variety Ettersburg No. 121 the brooming or bushy character of the disease is much more pronounced than in the foregoing and other varieties. The characteristics of the disease, then, are dwarfing of the plant, spindliness of petioles and an arching downward of the margins of leaflets, which are lighter in colour than in normal plants. The runners are much shortened, resulting in the formation of the young plants close to the parent (99).

Host Range and Distribution. The virus has only been recorded on strawberry plants; the following American varieties have been found affected: Marshall, Nick Ohmer, Oregon and Ettersburg. The disease has so far been found only in Western Oregon, U.S.A.

FRAGARIA VIRUS 4. Plakidas

Synonyms. *Strawberry Dwarf Virus*, Plakidas, 1928; *Strawberry Virus 3*, J. Johnson's classification.

The Virus and its Transmission. There is no information on the properties of the virus which does not appear to be sap-transmissible. Plakidas (74) suggests that *Aphis forbesi* Weed. may be the insect vector.

Disease Caused by Fragaria Virus 4

Rosaceæ

Fragaria vesca. Strawberry. *Dwarf disease.* The disease caused by this virus has been called "dwarf" by Plakidas, it is also known locally as "wild plant" and "white bud." The leaves of affected plants are much reduced and strikingly deformed,

with short petioles and somewhat elongated, asymmetrical, crinkled leaflets. The older diseased leaves are slightly greener and more shiny than healthy ones. The petioles, veins, and under side of the young leaflets are often reddish-purple. Affected leaves are abnormally brittle. Diseased plants may become "blind," i.e., the main bud is killed. This usually causes the death of the plant, but occasionally several adventitious buds may develop from the crown. The leaves developing from these secondary buds are small with long spindling petioles. The root system appears to be unaffected. Chlorosis of the leaf is absent in this disease, and therein lies a difference from yellow-edge (*Fragaria Virus 1*).

Histopathology. Cytological examination of the pericyclic region of the young roots of dwarf strawberry plants shows many cells with marked signs of degeneration and containing one or more black-staining, amorphous bodies. The nuclei of these cells are almost invariably devoid of nucleoli, the membrane appearing to be either practically empty or containing a small quantity of granular, dark-staining material (74).

Geographical Distribution. The dwarf disease has so far only been recorded from the southern United States of America, particularly Louisiana.

Control of Strawberry Virus Diseases

There are three main avenues of approach to the question of control : firstly, the elimination of the insect vector ; secondly, the systematic eradication by "roguing" of diseased plants ; and thirdly, the production of resistant varieties.

Since the insect vector is the same for several diseases the same control measures will be applicable. A spray or dust containing nicotine is the best insecticide to use. In New Zealand the method recommended for the destruction of the strawberry aphis consists in cutting off the leaves of the plants in autumn with a scythe, care being taken not to injure the crowns. The straw which has been used in the summer to protect the fruit should be teased up lightly and, together with the cut leaves, should be fired from one side on a day when a good cross wind is blowing so that the fire will traverse the whole bed quickly. Provided there is only a light covering of rubbish, the fire will sweep swiftly across without damaging the crowns. This destroys all the aphides which would otherwise overwinter. An alternative method is to cut off all the leaves, rake them into heaps with the straw and burn at once. If the infestation has been very severe, the denuded

crowns should be sprayed with nicotine sulphate, 1 part to 800 parts of water plus about 3 to 4 lb. soft soap per 100 gallons of spray. *Aphis*-infested runners may be cleared by immersion in hot water for twenty minutes at a uniform temperature of 110° F. Only vigorous and strongly rooted plants should be so treated, and they should be planted immediately after treatment (22).

The success of attempted control of strawberry virus diseases by roguing runner beds largely depends (1) on obtaining a virus-free nucleus of runners with which to build up the runner beds, and (2) on the degree of accuracy with which all subsequently infected plants are identified and eliminated. The latter can only be achieved by avoiding varieties with high symptom-masking capacity such as Lefebre, by reducing as far as possible the masking of symptoms due to mite and insect infestation and by carrying out final inspections during the optimum (for England) period of symptom manifestation, *i.e.*, the latter half of September and October (32). The chief obstacles in the way of successful roguing are the almost complete masking of symptoms under certain conditions and the difficulty of recognising the diseases, especially "yellow-edge," in the "mild" form.

As regards "crinkle," the practicability of selection and roguing from the Marshall variety has been demonstrated, and has led to the certification of strawberry plants in the State of Washington. In the selection of plants and roguing for this certified stock the purpose was to eliminate all abnormal plants.

The development of resistant varieties of strawberries is promising because of the large number of commercial (American) varieties which show a marked degree of resistance and because the strawberry is a convenient plant for breeding since it is vegetatively propagated (73).

Virus Diseases of the Raspberry (*Rubus Idaeus L.* and *R. occidentalis L.*) and the Blackberry (*R. fruticosus L.*)

The raspberry is subject to infection with several virus diseases, some of which are of the mosaic type while others produce leaf distortion or necrosis. Bennett (10) considers that the problem of identification and classification of the raspberry viruses is complex and that not enough information is available to present a final verdict as to the number of distinct viruses involved. In a recent paper Cooley (21) presents evidence that only two viruses are concerned in the production of raspberry mosaic diseases in North America. He suggests the name "green mosaic" for the

terms previously used, *i.e.*, "red-raspberry mosaic" and "red mosaic," but leaves the term "yellow mosaic" unchanged. In the account of the raspberry virus diseases given here it has been decided to deal with them in the following manner. First, green mosaic or red-raspberry mosaic as it used to be called (*Rubus Virus 1*) and yellow mosaic (*Rubus Virus 2*) are described. These are the common mosaic diseases of raspberries in North America. Then the mosaic diseases of raspberries in Europe and especially England are dealt with separately under the heading of the three types of leaf symptoms as described by Harris (33). In the communication referred to these European mosaic diseases are not assigned to specific viruses. Following upon these, the two leaf-curl diseases, alpha and beta (*Rubus Viruses 3 and 3A*), as described by Bennett, the streak disease (*Rubus Virus 4*), and the dwarf disease of blackberry (*Rubus Virus 5*) are discussed.

RUBUS VIRUS 1. Rankin and Hockey

Synonyms. *Red-Raspberry Mosaic Virus*, Rankin and Hockey, 1922; *Red Mosaic Virus*; *Raspberry Green Mosaic Virus*, Cooley, 1936; *Raspberry Virus 2*, J. Johnson's classification.

The Virus and its Transmission. Attempts to transmit the virus of green mosaic by means other than by grafting and by the agency of aphides have been unsuccessful. There appear to be three species of aphides which are capable of transmitting the virus, of which *Amphorophora rubi* is the most efficient vector. The other two species, *Amphorophora rubicola* and *A. sensoriata*, are of less importance, probably because the former is of limited occurrence and because the latter feeds on the more woody parts of the stems. These three aphides are described on pp. 505-509. The virus of green mosaic is rapidly lost by the vector *A. rubi*. Bennett (10) states that the aphis rarely transmits the virus beyond the second series in successive transfers and he considers that the insect is probably only a mechanical vector. There appears to be no information on the physical properties of the virus of green mosaic (*Rubus Virus 1*).

FIG. 19.

(Upper) *Fragaria Virus 3* (causing Strawberry Witch's Broom Disease). (After Zeller.)
 (Lower) *Rubus Virus 1* (red-raspberry mosaic virus) on black raspberry, var. Cumberland. (After Bennett.)



Diseases Caused by Rubus Virus 1

Rosaceæ

Rubus Idaeus, the red raspberry, and *R. occidentalis*, the American black raspberry. *Green mosaic*. The "green mosaic" disease is considered here as due to one virus entity only, but one which differs in severity and the symptoms on certain well-known American raspberry varieties are described.

Mild, var. *Cumberland* (black raspberry). Mottling mild, but distinct on first leaves of fruiting canes in the early spring. No symptoms visible on foliage produced later in the season; plants not stunted (see Fig. 19).

Var. *King* (red raspberry). Mottling very mild or not evident, restricted to first leaves of fruiting canes; plants not dwarfed.

Medium Severe, var. *Cumberland*. Mottling distinct on first leaves of fruiting canes, leaves sometimes distorted. Leaf petioles and cane tips necrotic in recently infected plants. Necrosis slight in plants in the second year with disease. Rings of mottled leaves often found on first-year canes marking periods of low temperatures; plants somewhat stunted.

Var. *King*. Mottling consists mostly of faded blotches, mild but clearly evident on the first leaves of the fruiting canes; plants not dwarfed (see Fig. 20 A.)

Severe, var. *Cumberland*. Mottling not excessive, leaves often deeper green than normal; leaf petioles and cane tips necrotic and brittle. Canes short and rosetted, sometimes producing normal shoots during periods of high temperatures; plants very much dwarfed, killed in two or three years.

Var. *King*. Mottling conspicuous on leaves formed at low temperatures and masked on those formed at higher temperatures. Plants little or not at all stunted (10).

Var. *Plum Farmer* (black raspberry). Within two weeks after infection with the virus of green mosaic, symptoms may develop in the tips of new canes. Small water-soaked areas appear in the portion of the cane on which the new leaves are expanding and enlarging. Similar discoloured areas develop on the petioles of the enlarging leaves. The tip of the cane usually makes no further growth and it becomes variously distorted. The water-soaked areas in the meantime become more extensive and turn to a deep blue colour. All of the tips of a small plant usually die during the same season and the plant either succumbs the following spring or persists as a dwarf with short rosetted new canes for a season or two. In the year following infection, most plants show

a less severe reaction. During the first stages of development of the fruiting laterals growth is normal, but towards the tip the internodes are foreshortened. In some plants the tips of the fruiting laterals are a tight rosette of leaves and fruit. In *Plum Farmer* the leaves of the fruiting laterals show a type of mottling characteristic of green mosaic in this variety. The yellow areas vary from a small dot-like mild mosaic to larger types. They are irregularly scattered and may show no relation to the veins. This type of mottling is constant enough to make it one of the most dependable characteristics for recognising green mosaic in the variety *Plum Farmer* (75). This disease is characterised by a low temperature maximum for symptom expression, by mottling, and by necrosis of the leaf petioles and cane tips. It varies in severity from a very mild mottling on the first leaves produced in spring, to a type which causes severe necrosis, accompanied by rosetting and resulting in the death of black raspberry plants in a few years. Retarded foliage development on the fruiting canes in spring is also a symptom of this disease (20).

RUBUS VIRUS 2. Bennett

Synonyms. *Raspberry Mosaic Virus 2*, Rankin, 1931; *Raspberry Yellow Mosaic Virus*, Bennett, 1927; *Raspberry Virus 2B*, J. Johnson's classification.

The Virus and its Transmission. *Rubus Virus 2* (yellow mosaic virus) is apparently not sap-transmissible and the insect vectors are the same as for *Rubus Virus 1* (green mosaic virus). The aphid, *A. rubi*, seems to retain this virus slightly longer than it does *Rubus Virus 1*. The physical and other properties of *Rubus Virus 2* are not known.

Diseases Caused by Rubus Virus 2

Rosaceæ

Rubus Idaeus. Raspberry. *Yellow mosaic*. The appearance of yellow mosaic is described on the varieties *Cumberland* and *King* for comparison with green mosaic caused by *Rubus Virus 1* on the same varieties.

Var. *Cumberland*. Plants more or less stunted and decidedly yellow in appearance. Leaves yellow, veins often lighter in colour, symptoms marked during most or all of the season, mottling not conspicuous or of a very coarse pattern. Cane tips and petioles not necrotic.

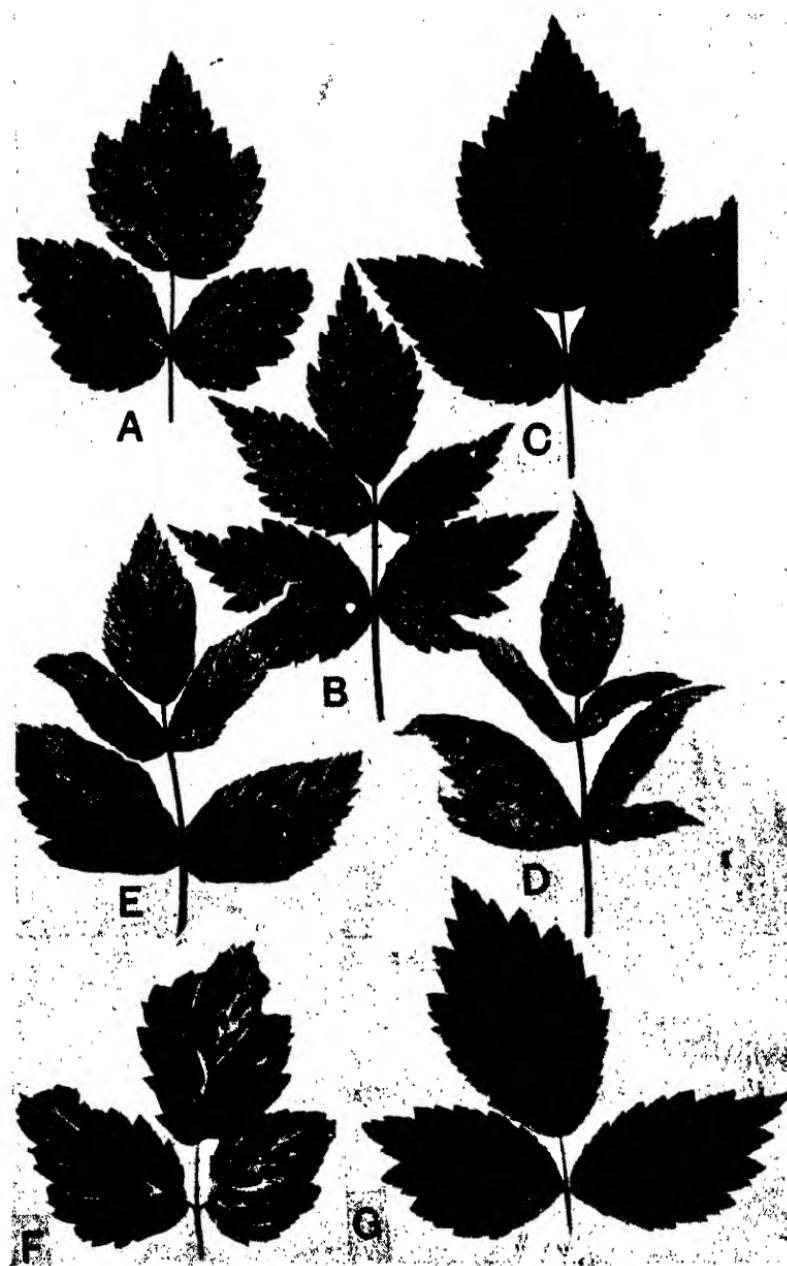
Var. King. Plants usually stunted and yellow. Leaves with yellowish cast, leaflets rolled downwards and inwards, mottling slight or of a very coarse pattern. Symptoms not masked by summer temperatures. This disease has a high temperature maximum for symptom expression and produces yellowing with little or no necrosis. On black raspberries the yellow mosaic virus causes less dwarfing than the severe forms of green mosaic and more than the milder forms (see Fig. 20 E). According to Rankin (75), black raspberries affected with yellow mosaic are severely dwarfed with the leaflets longer and narrower than normal and variously distorted and curled. Most of the area of a leaflet is either whitish or yellow with a few areas of puckered green tissue intervening. All parts of the plant are brittle as in green mosaic due to the turgidity of the tissue. Bennett (10), on the other hand, was unable to produce green blisters and brittle tissue in black raspberries with the yellow mosaic virus, and considers that Rankin was probably describing the symptoms due to infection with a mixture of the yellow and the green mosaic viruses. Disease complexes of this type frequently occur, and the disease they cause is described in a subsequent paragraph.

Disease Symptoms in Raspberries Caused by Double Infection with *Rubus* Viruses 1 and 2

Red raspberry plants of the variety King in south-west Michigan sometimes exhibit yellowing and stunting of the whole plant together with yellow leaflets which are also cupped and distinctly mottled. In black raspberry plants also, a disease occurs in which

FIG. 20. Raspberry, var. King, inoculated 1927,
photographed 1928.

- A. Lower leaf of first year cane from plant infected with *Rubus Virus 1* (red-raspberry mosaic virus).
- B, C, D. Leaves from first year cane inoculated with *Rubus Viruses 1* and *2* (red-raspberry mosaic and yellow mosaic viruses).
- D shows the mottling induced by the mixture of the two viruses at temperatures too high for *Rubus Virus 1* alone to produce symptoms.
- E. Leaf from the tip of a first year cane from a plant inoculated with *Rubus Virus 2*.
- F. Leaf from raspberry, var. Cumberland, infected with *Rubus Viruses 1* and *2*.
- G. Leaf from raspberry, var. Cumberland, infected with *Rubus Virus 2*. (After Bennett.)



the plants and leaves are yellow, the leaflets mottled, the cane tips necrotic and brittle, and the stems often striped with dark irregular reddish splotches. In both the foregoing cases, the symptoms are thought to be due to a double virus infection. It appears, also, as if the combination of the yellow and green mosaic viruses induces a greater amount of mottling even at temperatures high enough to suppress mottling in plants affected with green mosaic alone. This seems to indicate that the green mosaic virus is able to modify the symptoms of yellow mosaic on leaves developed at temperatures too high for green mosaic alone to produce any effect. Bennett (10) gives the following comparative description of the chief symptoms produced on red raspberry plants (var. *King*) by infection with yellow mosaic (*Rubus Virus 2*), green mosaic (*Rubus Virus 1*) and a mixture of the two. In yellow mosaic, the leaves of the fruiting canes are slightly stunted and yellow, the leaflets are rolled downwards and inwards, but are not appreciably mottled. On new shoots about 18 inches high, the lower leaves are normal and the tip leaves have the leaflets yellow, with the edges rolled downward and inwards. In green mosaic, the first leaves of the fruiting canes are distinctly mottled with light-coloured splotches, later-produced leaves appear normal. On the new shoots the lower leaves are distinctly mottled as on the fruiting canes, leaves produced later in the season are normal. Where the plant is infected with both viruses, all leaves on the fruiting canes are stunted, yellow and mottled and the leaflets are rolled downward and inwards. On the new shoots the lower leaves exhibit light-coloured splotches, with a ring of normal leaves above. The tip leaves are yellow, stunted, and mottled and the edges of the leaflets roll downwards and inwards. The cane striping appears to be confined to black varieties of raspberries affected with the combination disease (see Fig. 20).

Leaf Symptoms of Raspberry Mosaic in England

Three main types of mottling occur on the raspberry plant in England. Harris (88) refers to these as symptom types A, B and C, and regards them simply as a convenient mode of describing the range of mosaic leaf symptoms observed.

Type A Symptoms on Mitchell's Seedling. Chlorotic mottlings of varying size and irregular shape are evenly distributed over the entire leaf in contrast to type B, the individual chlorotic patches are not appreciably sunken and the leaf is not curled or distorted, but is quite normal in appearance except for the mottling

(compare types B and C). This type also occurs on Bath's Perfection and other varieties. A similar evenly distributed mottling, unaccompanied by curling or distortion, has commonly been observed on the lower leaves of the new growth of virus-free canes and is probably brought about by shading or other purely local factors. Such mottling can be differentiated from the type A mosaic by the fact that it appears on the lower or older leaves only whereas the mosaic mottling symptoms occur also on the younger leaves.

Type B Symptoms on Baumforth's Seedling B. In this variety the chlorotic areas or mottling vary in colour from pale green to greenish-yellow, and in size from minute specks to patches about 2 mm. across, not clearly delimited from the dark green areas. They are only slightly sunken, and transverse sections made through these areas indicate that there is a gradual and very slight decrease in thickness of the lamina in passing from the green to the chlorotic areas, due to a slight decrease in the depth of the palisade layer of cells. The dark green areas between the chlorotic areas are slightly convex when seen from above. The chlorotic areas are never evenly distributed over individual leaflets, but are generally concentrated between the main veins, giving the leaf a slightly striped appearance, and often they are more crowded towards the margins. This mottling is accompanied by a symmetrical downward curling of the leaflets about the mid-rib and sometimes by a downward curling of the mid-rib itself. One of the most characteristic features is the varied degree of mottling and curling of each of the leaflets composing a single leaf. A further distinguishing feature is the complete masking or suppression of these symptoms on leaves produced under hot summer conditions.

Type C Symptoms on Baumforth's Seedling B. This type of mottling can be differentiated from the B type on Baumforth's Seedling B by the following characters. The individual chlorotic spots are bright yellow to greenish-yellow and are more sharply defined and easily distinguished than those of the type B. Each spot is irregular in shape and sharply angular and about 2 mm. across, or more in advanced cases. Unlike the spots in type B they are deeply sunken or embossed, the leaf being thinner in these areas. These spots appear translucent and are generally evenly distributed over the leaf and individual spots commonly cut across a leaf vein or mid-rib. Spotting of this type is invariably accompanied by a curling of the laminæ, but in this

case the curling ultimately takes the form of an irregular or asymmetrical twisting, crumpling or distortion of the whole leaf. Symptoms of the C type are not masked under hot summer weather conditions.

The relation of the foregoing symptom types to the virus complex may be summarised as follows :—

Symptom type B. Virus 1 or Disease 1.

Symptom type A. Mild

Symptom type C. Severe } Virus 2 or Disease 2.

So far these diseases are only known to occur on the European red raspberry, but analogous symptoms occur on other species of *rubus* (e.g., loganberry and blackberry), and suggest that the host range may be wider.

Disease 1 is a simple mosaic, slow in its deteriorating action. Symptoms appear with regularity and are temporarily masked by hot, dry weather conditions.

Disease 2, as in the case of strawberry "crinkle," is clearly complex. A plant infected with the "mild" form does not appreciably deteriorate and the symptoms may be so slight as to be detectable only with difficulty. On plants artificially infected with this mosaic it has been impossible to detect symptoms during certain seasons, or the symptoms have been limited to a few scattered lesions on one or two leaflets. The serious phase arises apparently sporadically (probably by a further infection of "mild" cases), deterioration is extremely rapid, the plants soon cease to crop, and die out.

The two diseases and both phases of disease 2 have been transmitted by grafting, but no transition from one type of symptom to another has been observed, except from the "mild" to the "severe" phase of disease 2. Both diseases commonly occur on the same plant.

Relation of the English to the American Mosaic Diseases of the Raspberry. Harris (84) has carried out transfers of the American mosaic diseases to the two English index varieties of raspberry and obtained symptoms on the latter quite distinct from those normally occurring on these varieties in England. At the moment the weight of evidence indicates that the two disease-groups are not identical.

It is significant that the American mosaic diseases are transmitted by the aphid *Amphorophora rubi*, whereas numerous attempts to transmit the English mosaic diseases by means of the same aphid have consistently failed (Harris, 84).

RUBUS VIRUSES 3 and 3A. Melchers and Bennett

Synonyms. *Raspberry Leaf-curl Viruses Alpha and Beta*, Bennett, 1930; *Raspberry Yellows Virus*, Melchers, 1914; *Raspberry Viruses 1A and 1B*, J. Johnson's classification.

The Viruses and their Transmission. Some recent work by Bennett (9) has demonstrated the existence of two viruses of the



FIG. 21. *Rubus Virus 3* (Raspberry leaf-curl virus). Healthy raspberry plant (*left*), diseased plant (*right*). (After Berkeley.)

curl type producing similar or identical symptoms. These are named the alpha and beta curl viruses and are classified here as *Rubus Viruses 3* and *3A* respectively. The alpha curl virus is common in Michigan, U.S.A., on the Cuthbert variety of red raspberry, it is apparently only transmissible to red varieties of raspberries. The beta curl virus is common on the Cumberland variety of black raspberry in Michigan and Northern Ohio. This virus is transmissible to and from red, purple and black raspberries.

The leaf-curl viruses are not sap-inoculable and the insect vector is the aphid, *Aphis rubicola* (see p. 528), the winged form of which appears to play an important part in long-distance dissemination.

Diseases Caused by Rubus Viruses 3 and 3A

Rosaceæ

Rubus Idæus. Raspberry. *Curl Disease* (see Fig. 21).

Among American raspberries, the red variety Cuthbert and the purple and black varieties, Haymaker and Cumberland, are very susceptible to the curl disease. On these varieties, the characteristic symptoms consist of a curling of the leaves accompanied by the production of a deep green foliage and by a stunting of the entire plant. The curling of the leaves seems to be due to retardation in growth rate of the veins, while the intervening tissue continues to grow at a more or less normal rate, resulting in the apparent sinking of the veins, a downward curling of the leaf margins, and the production of folds or crinkles in the other parts. The leaves and canes of curled plants appear dry and do not wilt so readily as normal plants. By late summer, the leaves have usually taken on a bronze colour, thus giving the entire plant a coloration in contrast to the lighter green foliage of surrounding healthy plants. The degree of leaf rolling varies somewhat with the character of the soil on which the plants are growing and with the rate of growth. Slow-growing plants usually show very marked symptoms of curl, while rapidly growing plants on fertile soil may show practically no curling of the leaves and very little stunting of the canes. In all cases, however, there is a characteristic greasy appearance of the leaf surface. Symptoms of curl resulting from current season infection often appear on the tips of the new canes by July 1st; in the season following infection all parts of the plants show symptoms. Diseased canes lack hardiness and are subject to winter killing. Fruiting canes produce very short fruiting shoots and the berries are small and of poor quality (8).

It is doubtful if the American leaf-curl viruses of the raspberry occur in England. When such viruses are transmitted by grafting to the English varieties, Lloyd George and Baumforth's Seedling B, severe symptoms quite different from any observed in England are produced, and these are followed by local necroses of laterals and the death of the whole plant (Harris, 84).

RUBUS VIRUS 4. Wilcox

Synonyms. *Raspberry Eastern Blue Stem Virus*, Wilcox, 1922; *Raspberry Streak Virus*, Zeller, 1923; *Raspberry Rosette Virus*, Rankin, 1923; *Raspberry Severe Streak Virus*, Rankin, 1931; *Raspberry Leaf-curl Virus 3*, Rankin, 1931; *Raspberry Virus 3*, J. Johnson's classification.

The Virus and its Transmission. The virus does not appear to be sap-inoculable and there is no information on its insect vector or physical properties.

Disease Caused by Rubus Virus 4**Rosaceæ**

Rubus Idæus. Raspberry. *Streak disease*. Diseased plants present an appearance which suggests mild leaf-curl symptoms. Plants are stunted after the first year and become smaller each succeeding season. Leaves on such plants are nearly always distinctly curled and usually placed close together on the canes. The foliage is a shade darker green than normal. Leaves, especially those at the tip of the canes, have a peculiar twist of the mid-rib, with a decided tendency to bend at an angle, and recurve in such a way as to bring uppermost the dorsal side of the tips of the leaflets. As observed in Michigan, mottling is not a marked characteristic of streak. Bennett considers that there is probably much variation in this symptom under different conditions of temperature and with different varieties. Wilcox (91), in Ohio, and Zeller (97), in Oregon, describe a uniform mottling frequently associated with streak.

Streaking or striping of the stem of the new canes is a fairly constant symptom of the disease. Bluish-violet, discoloured areas which may consist of dots, spots or longitudinal stripes, appear on the main stems and laterals. The blue markings are usually most abundant near the bases, but also occur on the branches of the new canes or on the fruiting spurs (8).

Rankin (75) describes a disease which he designates mild streak in distinction to the streak described above which he calls severe streak. Rankin considers that mild streak is due to a distinct virus. The symptoms are similar to those of severe streak, but are less pronounced, nevertheless the disease is considered to be of importance because of its effect on the fruit which becomes dry, "seedy" and crumbling.

RUBUS VIRUS 5. Zeller

Synonyms. *Blackberry or Loganberry Dwarf Virus*, Zeller, 1927; *Raspberry Virus 2D*, J. Johnson's classification.

The Virus and its Transmission. *Rubus Virus 5* is not transmissible by sap-inoculation and the insect vector appears to be the aphid *Capitophorus tetrarhodus*. There is no information on the physical properties of the virus.

Disease Caused by Rubus Virus 5

Rosaceæ

Rubus fruticosus L. Blackberry, loganberry. *Dwarf disease*.

The progress of the dwarf disease in the plant is characterised by distinct morphological and physiological changes, most marked in the leaves and stems, and to a certain extent in the fruit. These consist principally of an abnormal colour accompanied by malformation and general dwarfing. The symptoms vary somewhat according to the age of the plants at the time of infection, but the general characters are the same in all stages of growth after the initial year of infection. In a mature plant infected in the field, the main symptoms are in the leaves, which are smaller than normal and of an obovate shape. New canes produced on such a plant in the second season show the typical foreshortening of the stems with spindling growth.

In young plants there is a certain amount of necrosis along the veins and particularly in the mesophyll between the veins where the virus-bearing aphides have fed. Following this necrosis, all growth of the current season produces crinkled leaves with irregular margins and a chlorotic mottling. The most usual type of mottling is a finely netted spotting which follows rather regularly the netting of the finer veins of the leaf.

The stems of diseased plants are not mottled or streaked and the colour appears to be normal. The spindliness of the canes and the shortening of the internodes are the chief characteristics of plants in the early stages of the disease. Later the canes are extremely foreshortened, characteristically stout and stiff with very short internodes.

The fruit develops to a fair size, but there is a tendency for the druplets to fall from the receptacle.

Geographical Distribution. So far as is known, the geographical distribution of the dwarf disease is limited to various localities in California where the loganberry and "Phenomenal" black-

berry are grown, to the coastal slope, Willamette and Umpqua valleys in Oregon, and the portion of Washington west of the Cascade mountains (98).

Symptoms in Raspberries Due to Mixed Virus Infections

Two or three of the viruses described above are liable to attack the same raspberry plant, and symptoms of these combination diseases may often be observed under field conditions. The American variety Cuthbert, when infected with both mosaic and curl, exhibits symptoms of both diseases, but with the curl symptoms usually predominating. The leaves are typically curled and of a deeper green hue, but such leaves may show a high degree of mottling varying from faint yellowish spots to a condition of distinct deep green elevations or blisters surrounded by a lighter green or yellowish field. In the American variety Cumberland affected with the same virus complex, the curl symptoms are predominant and the mosaic mottling so faint as to be easily overlooked. Plants infected with a combination of yellow and green mosaic show the typical yellow cast of yellow mosaic in the recently infected shoots, but with additional mottling. The tips of the canes show the external and internal discolorations characteristic of green mosaic and the brittleness of the tip and leaf petioles (8).

Geographical Distribution. The virus diseases of the raspberry are widely spread throughout Canada and the United States of America. In Washington green mosaic seems to be the most prevalent and destructive, both red and black raspberries being equally affected. In the raspberry-growing sections of Eastern Canada both types of mosaic are important and leaf-curl is also prevalent. In British Columbia, however, there appears to be little mosaic disease and the spread is very slow. The streak disease of raspberries is prevalent in the U.S.A., in Ohio and Illinois, and has been found to a limited extent in Wisconsin, Michigan and New York.

In Great Britain mosaic is widely spread throughout the country, though leaf-curl has not been observed.

Methods for the Control of Raspberry Virus Diseases. In discussing the methods for the control of the raspberry virus diseases, it is proposed to describe shortly the procedure which is current in various countries. In the U.S.A. the nursery law of New York requires that all raspberry nursery stock offered for sale must come from fields inspected and rogued for virus diseases.

It seems probable that the spread of virus diseases of bramble fruits is much too slow often to cause serious loss during the life of a single planting provided the stock is clean at the beginning. The key to the control situation then lies in establishing sources of disease-free stock and continuing these sources in spite of the frequent introduction of virus diseases from the wild species of *Rubus* which are prevalent in waste spaces over the greater part of the U.S.A. Roguing is most effective in the first year of a planting. If the plants are examined in the latter part of June, usually the symptoms of all virus diseases can be seen. As a rule aphides have not become numerous in such young fields and the plants are small enough to be removed from the field without much added effort. It is perhaps worth emphasising that plants dug in roguing should be lifted as carefully as possible to avoid shaking off the aphides (8). It should also be remembered that wild brambles are frequently a source of infection.

In Canada it has been found that the use of healthy certified raspberry stocks has been in every respect satisfactory. Where such stock has been set out and has been carefully inspected and diseased bushes rogued during the growing season, the percentage of infection has been very low. Although certification of stock is considered to be the first essential in control, the roguing of diseased bushes during the first two seasons at least should by no means be neglected. In the Eastern Provinces and States of Canada the campaign against virus diseases of the raspberry aims first at the improvement of the stocks of cane of existing commercial varieties by the elimination of mosaic-cane from the sources of cane supply by a system of inspection and certification, and, secondly, in the breeding of new varieties resistant to mosaic infection. In Canada the canes are largely propagated in special non-fruтиng nursery rows, and the inspection is now limited to such beds. In the United States, however, the propagation of cane in fruiting plantations is still the custom and the inspections are carried out on such plantations.

As regards the production of varieties which are resistant to virus diseases, long-range systematic research in raspberry breeding is in progress in Canada and the U.S.A., and the object in mind is the combination by systematic breeding of desirable horticultural characters with resistance to virus diseases. The Vineland Horticultural Experiment Station, Ontario, has produced the Viking variety which combines high cropping and fruit qualities with winter hardiness greater than that of the Cuthbert variety.

American breeders aim at producing varieties combining *low infectability*, by the insect vectors, with *high susceptibility and symptom expression*, i.e., plants which rarely become infected, but when infected can readily be eliminated. *Viking* is a good example of one of the earlier produced varieties in this category. More recently the New York station at Geneva has produced a range of notable varieties of this type, including *Newburg* and *Taylor*.

It is significant in view of the probable distinction between the English and American raspberry mosaic diseases, that the best results at Geneva, New York, have been produced by crossing American varieties with the English *Lloyd George* variety.

In England experiments on commercial control measures have, up to the present time, been mainly concentrated on the widely grown *Lloyd George* variety, and the conclusion has been arrived at that the direct control of raspberry mosaic by the removal or roguing of infected stools in established fruiting plantations is impracticable, the labour involved being out of proportion to the immediate benefit obtained. It is considered that a practicable scheme of control should aim at providing the grower, at any stage in the life of a variety, with virus-free supplies of cane for planting up his fruiting plantations. Further it is recommended that although the roguing of commercial fruiting plantations is impracticable, a satisfactory control can be achieved by a system of "cane-nurseries" established solely as a source of supply of cane free from mosaic infection, for the planting up of commercial plantations. The success and reliability of a nursery largely depends on the accurate identification of infected stools and on the immediate removal and destruction of such stools. The best times for such inspections are in early July and in early September, and it is important that any canes showing mosaic should be dug up and burned, together with those adjoining on either side (86).

HOLODISCUS VIRUS 1. Zeller

Synonym. *Holodiscus Witch's Broom Virus*, Zeller, 1931.

The Virus and its Transmission. The virus is transmissible by grafting, but probably not by sap-inoculation. The insect vector is thought to be the *aphis, Aphis spireae* Schout.

Disease Caused by Holodiscus Virus 1

Rosaceæ

Holodiscus discolor Max. "Ocean Spray." *Witch's Broom*. When the disease first makes its appearance on a plant the new

lateral branches from an old stem are very slender and wirelike with rather short internodes and small leaves. As a rule there are two or three of these slender laterals from each node, while in healthy plants the laterals are thicker and there is only one per node. In the second or third year there is considerable multiplication of the laterals from each node on stems more than two years old and these laterals are much branched in contrast to the laterals of healthy plants. New canes, which arise from or near the crown after the plants become affected, are short and give a stiff appearance. In these canes the internodes are short; the main stems have little tendency to branch so there are usually no blossom clusters. There are several buds at each node and these produce very short spindly laterals.

The leaves of affected plants are very small and crowded, giving the canes a leafy appearance. Where they are not shaded they turn a bronzy red early in the summer. This general reddish tone may appear early in June when the spring is cool. The colour stands out in contrast to the bright green of neighbouring healthy plants (100).

Host Range. Attempts to transmit the virus to the following related rosaceous plants were unsuccessful: *Spiraea thunbergii* Sieb., *S. Vanhouttei* Zabel, *S. prunifolia* S. and Z., *S. Douglasii* Hook, and *Physocarpus capitatus* Ktze.

Geographical Distribution. The disease was first observed in 1925, and has so far only been recorded from the western slope of the Cascade Mountains, Oregon, and from Thurston County, Washington, U.S.A.

PRUNUS VIRUS 1. Kunkel

Synonyms. *Peach Yellows*, E. F. Smith, 1888; *Peach Yellows Virus*, Kunkel, 1933; *Peach Virus 1*, J. Johnson's classification.

The Virus and its Transmission. The virus of peach yellows is not transmissible by mechanical inoculation with expressed sap. The insect vector has recently been shown by Kunkel to be a leaf-hopper, *Macropsis trimaculata* Fitch (see p. 484) (51). There seems to be a delay in the development of infective power within this leafhopper ranging from ten to twenty-six days with an average period of sixteen days. In one instance the minimum period has been as short as seven or eight days. Hartzell (42) has recently reported the occurrence of X-bodies, similar in appearance to those occurring in affected peach tissue, within the

intestinal walls and salivary glands of virus-infected leafhoppers. This is the first record of this type of inclusion in an insect vector.

At present there is no information on the physical or other properties of this virus and the question of seed transmission is still an open one.

Diseases Caused by Prunus Virus 1

Rosaceæ

Prunus persica (L.) Stokes. The peach. *Peach yellows*. The foliage of affected trees is usually paler than that of normal trees and there is a tendency for the leaves to fold together lengthwise along the mid-rib in a V-shaped manner; such leaves are rather turgid and brittle. In young trees one of the first symptoms to be noticed is the failure of the latent buds to remain dormant. These unfold into yellowish leaves scarcely more than an inch in length giving the tree a bushy appearance. The larger leaves may be mottled with areas of dark and light green. Another early symptom is a clearing of the veins in leaves near the tips of young branches. In older trees the leaves have a tendency to curl slightly and droop, and are of a yellowish-bronze colour in contrast with the normal green foliage of healthy leaves. The symptoms become more pronounced as the disease progresses and the leaves show a claw-like curling. Premature unfolding of leaf buds is another characteristic of the disease. These develop into willowed shoots in which the terminal buds are not dormant, resulting in a wiry broom-like growth bearing very narrow yellowish leaves which are often spotted with red and continue to grow after the fall of the normal leaves. Twigs arising from the lower side of a branch, and shoots on branch terminals, have a tendency to grow vertically. The production of numerous thin upright-growing shoots bearing small slightly chlorotic leaves is a conspicuous symptom of the yellows disease after it is well established (see Fig. 22).

The fruit ripens prematurely and shows definite symptoms of disease. The skin of the peach is highly coloured, spotted with red or purple, and the flesh marbled with crimson with pronounced colouring round the pit.

The fruit is apt to be large, but is of an inferior quality with a bitter taste. Such premature fruit may either all appear on a single branch with the rest of the fruit apparently normal, or the normal fruit may be on a single branch with the rest of the fruit premature.

In the second year of infection, the fruit is apt to be smaller than normal.

Affected trees set their buds earlier in the autumn so that these are well developed when winter comes. In the spring both the flower buds and leaf buds develop earlier than in healthy trees. This is especially noticeable when a single branch of a tree is



FIG. 22. Three peach seedlings of the same age. The plant on the left shows typical symptoms of infection with *Prunus Virus 2* (peach rosette), that in the centre is infected with *Prunus Virus 1* (peach yellows), and that on the right is healthy. (After Kunkel.)

diseased. The affected branch will be in full bloom while the fruit buds in the remainder of the tree are undeveloped. It may be mentioned here in parenthesis that cutting out such an affected branch does not arrest the systemic spread of the virus. Occasionally diseased trees blossom in the autumn.

The following are the most characteristic symptoms of peach yellows, the presence of "witches' brooms," the premature ripening of the fruit from a few days to three weeks early, the premature unfolding of leaf buds and the tendency of the terminal shoots of

larger limbs to grow vertically. These shoots are of a slender wiry character (41). Intracellular inclusions, or X-bodies, are present in the cells of petiole, blossom and root-hair tissues.

Prunus domestica L. The plum. The plum is susceptible to infection with the virus of peach yellows and is an important host for two reasons. First, it is a favourite host for the insect vector which breeds upon it in large numbers, and, secondly, certain species of plum are symptomless carriers of the virus. The variety of the Japanese plum (*Prunus salicina*), Abundance, is such a carrier. On the other hand, another variety of the same species, Santa Rose, shows the marked inward rolling of the foliage characteristic of infection with the peach yellows virus.

Host Range. The virus has only been recorded as attacking the following: the peach, *Prunus persica* (L.) Stokes; the nectarine, *P. persica*, var. *nucipersica* Schneid.; the almond, *Prunus communis*, Fritsch; the apricot, *Prunus armeniaca* L.; the Japanese plum, *Prunus salicina* Lindl.; and the cultivated plum, *Prunus domestica* L.

Geographical Distribution. Peach yellows has been known as a distinct disease for about 150 years, and it is confined to eastern temperate North America. The disease is prevalent in south-eastern Pennsylvania, New Jersey and Delaware. It was first observed near Philadelphia in 1791, and was reported from New York during the first decade of the nineteenth century, from Connecticut in 1814, from Massachusetts in 1854, from Michigan in 1866, and from Ontario, Canada in 1878. The distribution of the disease has not markedly changed in the last fifty years. It is known to occur in Ohio, Indiana and Kentucky, and was reported from Illinois in 1927. From Virginia its range extends southwards along the Appalachian Mountains with occasional outbreaks in Tennessee and the Carolinas.

Sporadic outbreaks have occurred in Iowa, Missouri, Arkansas, Northern Texas and Nevada, but the disease has never become established west of the Mississippi River, nor have there been authentic reports from the extensive peach-growing districts of Georgia and California (41).

Control. The known distribution of the insect vector of peach yellows, *Macropsis trimaculata*, corresponds roughly with the known distribution of the disease. Measures for its control, therefore, must aim at the reduction in the numbers of the leaf-hoppers. An important factor which must be considered in any

efforts at control of peach yellows is the susceptibility of various species of plum to the disease. The wild plum, *Prunus americana*, the myrobalan plum, *P. myrobalan*, and the Japanese plum, *P. salicina*, are all liable to infection with the peach yellows virus, and the myrobalan plum also carries the virus with the minimum of symptoms. Plum trees, especially *P. salicina*, are the favourite host plants of the leafhopper, and as many as 10,000 individuals of this insect have been observed on a single tree of Japanese plum. Control measures against the leafhopper therefore must include the removal of the wild plum from the vicinity of peach orchards. Any spraying operations must be carried out equally on peach and plum trees (58).

Experimental cures of young peach trees affected with yellows have been obtained by subjecting the plants to high temperatures. Potted trees were incubated in a hot room at temperatures varying from 34° to 36° C. for two weeks or longer. Some difficulty was experienced in destroying the virus in the roots which, being in moist earth, do not reach the temperature to which the tops are exposed owing to evaporation (52).

Dormant trees can be cured by immersing them for about ten minutes in a tank of water held at 50° C. The trees are not seriously injured by this treatment. The yellows virus in buds can be inactivated by immersing bud sticks in water held at a number of different temperatures. At 34° to 35° C. the virus is inactivated in four to five days, at 38° C. in eleven hours, at 42° C. in forty minutes, at 46° C. in fifteen minutes. The bud tissues are able to endure longer treatments than are necessary to destroy the virus (54).

PRUNUS VIRUS 1A

Synonyms. *Little-Peach*, E. F. Smith, 1898; *Little-Peach Virus*, Bennett, 1926; *Peach Virus 3*, J. Johnson's classification.

The Virus and its Transmission. In a recent paper Kunkel (53) has demonstrated that peach trees affected with little-peach disease are immune from infection with the virus of peach yellows and *vice versa*. Since invasion of healthy peach trees by either virus protects them against the disease caused by the other, it is concluded that the two viruses are closely related, and that little peach is caused by a strain of the same virus causing peach yellows. The virus is similar in behaviour to the type virus and the insect vector is probably the same, *i.e.*, *Macropsis trimaculata* (see p. 484.).

Diseases Caused by *Prunus* Virus 1A

Rosaceæ

Prunus persica (L.) Stokes. Peach. *Little-peach disease* (see Fig. 23). The first visible symptom in trees infected with little-peach is distortion of the young leaves at the tips of affected branches, while reduction in the size of, and delayed ripening in, the

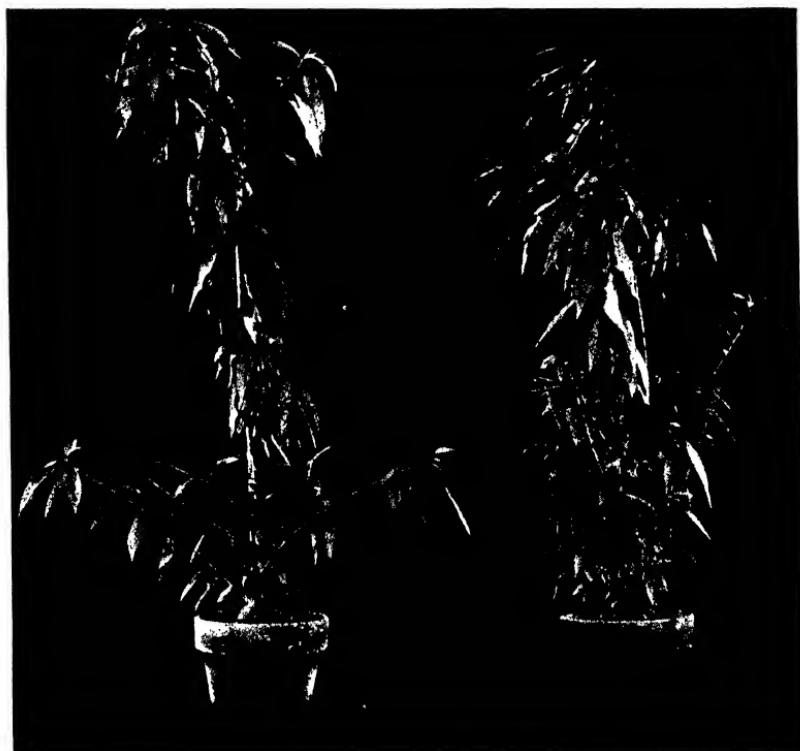


FIG. 23. *Prunus* Virus 1A (causing little-peach disease). Peach plant on left healthy, that on right diseased. (After Kunkel.)

fruit have long been considered the most outstanding symptoms of the little-peach disease. Bennett (7), however, considers that leaf and twig characters are more uniform than fruit symptoms. Affected trees are often stunted, with pale yellow leaves and a compact bushy appearance. The centres instead of being somewhat open are usually filled with shoots covered with clusters of leaves and short lateral branches. The terminal growth of the main limbs

in advanced cases is also of this general type. The ripening of the fruit may be delayed a few days or as long as three weeks. The fruits are reduced in size sometimes, but at other times are normal and ripen only a few days late. The flavour is usually poor, but fruits of good size and quality may be produced on affected trees. Observations made in 1931 by Cation (16) showed that in the initial stages of the disease the foliage is of a deeper green than normal. These large, dark green leaves near the terminal shoots show a drooping appearance and lie closely pressed to, and almost parallel with, the shoots from which they originate, instead of standing out perpendicularly from the branch as in a normal tree. A condition known as "feathering" or "fuzziness" often accompanies the disease; here short shoots and clusters of leaves spring from the basal portion of the main limbs. In cases where the disease is well established there may be a distinctive clustering, pallor and curling of the foliage. The downward curl of leaves on trees affected by little-peach consists of a bending of the mid-rib downwards and inwards, but the halves of the leaves do not usually fold together as in yellows. The petioles or stems of the leaves are often twisted and bent in such a manner as to disturb the normal arrangement of the leaves.

Prunus salicina, *P. simonii*. Japanese or Oriental plums.

Plum trees and particularly the Japanese plum (*Prunus salicina*) are susceptible to infection with the virus of little-peach. As in the case with peach yellows, certain varieties of plums carry the virus without symptoms while others show a marked inward rolling of the leaves. *P. salicina*, var. Chabot, and *P. munsoniana*, var. Chalco, are symptomless carriers of the virus, while *P. simonii*, var. Simon, and *P. salicina*, var. Satsuma, show visible symptoms when infected (58).

Control. The same methods of control are applicable to *Prunus Virus 1A* as are recommended for the type virus, and the virus yields to the same heat treatment.

PRUNUS VIRUS 2. McClintock

Synonyms. *Peach Rosette*, E. F. Smith, 1891; *Peach Rosette Virus*, McClintock, 1928; *Peach Virus 2*, J. Johnson's classification.

The Virus and its Transmission. *Prunus Virus 2* is not sap-transmissible and the insect vector is not known. The virus is readily transmitted by means of grafts or budding. There is no evidence of seed or soil transmission, nor information on the properties of the virus.

Diseases Caused by *Prunus* Virus 2

Rosaceæ

Prunus persica (L.) Stokes. The peach. *Peach rosette* (see Fig. 22). Peach rosette is more striking in its onset and more rapid in its progress than peach yellows. It frequently develops in the early spring and is more likely to appear all over the tree rather than on a part only as is the case with peach yellows. The chief symptom of peach rosette is the production of compact tufts or rosettes, 2 to 3 inches long, and containing several hundred leaves. At the base of these tufts are one or two abnormally long and straight leaves with inrolled margins. These outer leaves turn yellow and drop readily in early summer. An attacked tree nearly always dies the following autumn or winter. If only part of a tree is attacked that portion dies first and the remainder of the tree develops symptoms the following spring, dying in its turn in about six months. The colour of the foliage is yellowish-green. Trees affected by the rosette virus may show either of two distinct types of symptoms. They may wilt and die, or they may develop the characteristic rosette type of growth.

Peach trees affected with rosette either bear no fruit or, if borne, it shrivels and falls prematurely.

This disease differs from peach yellows in the following characteristics. There is no premature, red-spotted fruit, the compact bunching of the leaves is very characteristic and the disease is severe from the onset and progressive instead of chronic as with peach yellows (79). Since trees infected with rosette (*Prunus Virus 2*) are susceptible to infection with the viruses of yellows and little-peach (*Prunus Viruses 1* and *1A*), the rosette virus is evidently a distinct entity (53).

Prunus armeniaca L. The apricot. Apricot trees have been artificially infected with the virus of peach rosette. The chief effect lies in a marked stunting of the tree, but the shoots do not appear to have the typical rosetted appearance shown by the peach. The internodal growth, however, is much less than that of normal peaches. The leaves of affected apricots show a mosaic mottling and are of a yellowish tinge.

In addition to the apricot the following have been infected either naturally or experimentally with the virus of peach rosette; the cultivated plum and damson (*Prunus domestica* L.), the wild plum (*P. americana*), the cherry (*P. cerasus*), and the almond (*Prunus communis* Fritsch). On all these trees the symptoms are of the same general nature, rosettes of leaves with a yellowish pallor.

The variety Marianna plum appears to be immune from rosette (59).

Geographical Distribution. Peach rosette was first noted in Georgia, U.S.A., in 1881. By 1891 this disease had been reported from twenty-two counties in Georgia. Since 1903, reports of the presence of rosette have come to the Plant Disease Survey of the U.S. Dept. of Agriculture, from nineteen counties in Georgia, one county in Alabama, five counties in South Carolina, four counties in Tennessee, one county in West Virginia, nineteen counties in Missouri, and two counties in Oklahoma (59). There appear to be no records of this disease in Europe.

Control. Since the insect vector of peach rosette has not yet been identified, measures for the control of this disease are limited to a careful eradication of affected trees. In carrying out such eradication it must be borne in mind that the apricots, plum and damson and the almond are also susceptible to the disease. The heat treatment used by Kunkel (54) for the control of peach yellows is also applicable for peach rosette, but the rosette virus is somewhat more refractory and requires ten minutes at 50° C. for inactivation.

PRUNUS VIRUS 3. Hutchins

Synonyms. *Phony Peach*, Neal, 1921; *Phony Peach Virus*, Hutchins, 1930; *Peach Virus 4*, J. Johnson's classification.

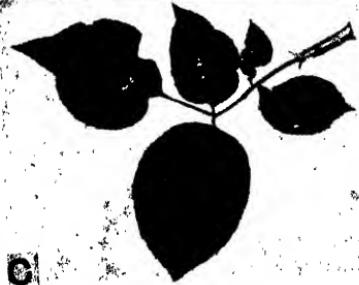
The Virus and its Transmission. The virus is not sap-transmissible, but can be transferred by root-grafting. The incubation period is long, two full seasons' growth ordinarily being required. The insect vector is not definitely known, but Hutchins suggests that the peach borer, *Egeria exitiosa* Say., may possibly be concerned in the spread of the phony virus, since it fulfils the conditions

FIG. 24.

- A. Peach trees infected with *Prunus Virus 3* (causing peach phony disease); diseased trees left, healthy tree right.
- B. Peach from tree infected with *Prunus Virus 5* (peach mosaic virus); note the irregularities of surface contour (apical view).
- C. Rose leaf, showing symptoms caused by *Rosa Virus 1* (rose mosaic virus).
- D. Rose leaf, showing symptoms caused by *Rosa Virus 3* (rose wilt virus); early stage, showing recurved leaflets.

(A and B, after Hutchins; C, after White; D, after Grieve.)

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necessary for the transmission of this rather unusual disease agent. The virus is unusual in that it is apparently confined to the roots of affected trees and is not systemically present in the rest of the tree. This is shown by the fact that the disease has never been transmitted by buds, scions or seeds from phony trees. On the other hand, the virus may be communicated to a normal tree through the medium of a living graft union between a phony root and the root system of the normal tree. Although, as mentioned above, healthy scions when grafted upon phony trees may take on the growth characters of the disease, the virus itself is not present. The following theory has been suggested to explain the production of abnormal growth in phony trees although the virus is present only in the roots. The fact that the infective virus of the phony disease seems to require living root cells for its activity and multiplication in the plant, appears to support the view that the sap may transport to the shoot a deleterious root-cell by-product, possibly in the nature of a toxin or a toxin-provoking principle. It is also possible that inactivated or altered virus may be present in, and contribute to, the toxicity of the sap-stream from infected roots (46).

Diseases Caused by Prunus Virus 3

Rosaceæ

Prunus persica (L.) Stokes. The peach. *Phony disease.* This disease has been studied by Hutchins and the following account is based on his descriptions. Affected peach trees develop shortened internodes, a large number of lateral twigs and flattened dark green leaves, giving the appearance of compact, dense growth with luxuriant foliage. Decided dwarfing results and is especially prominent in young trees. On the whole the foliage of a phony-diseased tree presents a striking depth and richness of green colour that is further enhanced by a vivid and unusual lustre. Actually the phony tree has fewer leaves than the normal ; this is probably due to the dwarfing effect of the disease, resulting in shorter twigs and fewer leaf buds per tree. Owing, however, to the closer concentration of the leaves and the increased number of leafy lateral branches there is a preponderance of exposure of flat upper leaf surfaces, thus giving an appearance of excess leafage. Unlike some of the virus diseases previously described, the phony disease usually develops simultaneously on all branches and in about equal intensity. Certain characters and behaviour exhibited by diseased trees at different seasons of the year have important

diagnostic value and are emphasised by Hutchins as follows : in *winter* phony trees are dwarfed and show short terminal and short lateral twigs, short internodes, and profuse lateral branching from growth that has been produced since the tree developed symptoms of the disease. In *spring* the rest period ends prematurely in diseased trees. For a given variety and under similar orchard conditions, the flower- and leaf-buds of a phony tree usually start growing a few days earlier than those of a normal tree. The phony tree will ordinarily pass the blooming period and show very advanced foliage before the normal trees have dropped their petals. Thus for a period of ten days or so the phony trees are easily identified by this character. In *summer* an additional diagnostic feature is afforded by the unpicked fruit at harvest time, this is decidedly smaller than the average for normal trees and ripens a few days earlier. In the *autumn*, phony trees tend to keep their leaves several days longer than healthy trees and may be in full foliage for a considerable time after the latter have become defoliated (see Fig. 24, A).

There is no characteristic symptom on the fruit, but individual fruits from phony trees are small and may be distinctly poorer in flavour than normal fruit, though better in colour. The ripening of diseased fruit of a given variety will begin a few days earlier than that of healthy fruit in the same orchard and may extend over a period of a week or ten days.

The characteristics of phony disease may be summed up as follows : affected trees display rich deep green leaves, smooth bark, and much dwarfed, but otherwise healthy appearing fruit. Dwarfing of the tree, the aspect of the tree in leaf, small fruit, crop reduction and certain marked seasonal behaviour, constitute the principal departures of the phony tree from the normal.

Laboratory Identification Test

Hutchins (46) describes the following confirmatory aid to the identification of phony disease. The test consists of immersing a transverse section of root in absolute methyl alcohol which has been acidified by the addition of a few drops of concentrated hydrochloric acid. After the lapse of three to five minutes the presence of phony disease is indicated by the appearance in the wood of numerous purplish spots. The spots may vary in size from pin points to areas about 2 mm. in diameter ; the number also may vary. For a positive test, from ten to fifty or more spots, very definite, intensely coloured and distributed over the entire

surface, must stand out in sharp contrast against a clear background of whitish or faintly purple wood.

In carrying out this test, pieces of root 4 to 6 inches long and $\frac{1}{2}$ to $\frac{3}{4}$ inch in diameter should be selected, taking one or more from each of the four sides of the tree. The roots should be washed and cut across, a transverse section about $\frac{1}{2}$ to 1 mm. in thickness is then cut with a sharp knife and immersed in the test reagent. Care must be taken that the root from which the section is cut is clean, white and free from any spots or blemishes which would obscure the test.

The test reagent is prepared by adding 1 to 5 drops of pure concentrated hydrochloric acid to each 25 c.c. of absolute methyl alcohol. The proper degree of acidity is important and varies somewhat with the specimens. The optimum acidity gives a positive reaction in three to five minutes. For the test it is best to use flat-bottomed watch glasses, each containing 5 c.c. of reagent, set out on a white background. If the acidity is too strong or the sections are allowed to remain too long in the reagent the entire surface of the sections, whether diseased or normal, may display a purple colour.

Host Range of *Prunus Virus 3*. There do not appear to be any records in the literature of natural infections of the phony disease on any host other than peach. Other species, when budded or grafted to phony peach trees develop the growth characters typical of the disease. This has been demonstrated in experiments where *Amygdalus davidiiana* and commercial varieties of almond, apricot and nectarine were grafted to phony peach trees. The dwarfing effect was particularly noticeable in *A. davidiiana*. Growth of this species when grafted on a normal peach tree was in some cases 10 feet in one season, as compared with a bushy, profusely branched growth of 18 inches on a phony tree (46).

Geographical Distribution. The disease was first observed about forty years ago in peach orchards near Marshallville, Georgia, U.S.A., but did not become sufficiently serious to cause alarm till 1915. Since that date more than a million trees have become infected in Georgia alone, and the disease has been recorded from Florida, Alabama, Mississippi, Tennessee and the Carolinas. It has crossed the Mississippi and the Ohio rivers to Louisiana, Texas, Arkansas, Oklahoma, Missouri and Illinois.

Control. At the moment the only method of control applicable to phony disease is the eradication and destruction of all trees showing signs of disease. It should be remembered that the wild

peach tree may also be a source of infection and must be included in any campaign of eradication. In districts where the phony disease is prevalent the wholesale destruction of all valueless peach trees in abandoned orchards and home orchards is strongly advised. Attempts are being made to limit the spread of phony disease by legislation, thus the importation of peaches, nectarines, etc., into Canada from the U.S.A. is forbidden unless accompanied by a certificate stating that the trees in question are free from phony disease and also that no phony disease was present within an area of one mile radius.

By means of intensive measures in the south-eastern states of North America the number of phony peach trees in the worst situations has been reduced from 177 per thousand in 1929 to less than twenty per thousand during the last two seasons (1935-36). During 1936 over two million abandoned, and 3½ million escaped peach trees have been eradicated in 129 counties of eleven States of the Union.

PRUNUS VIRUS 4. Bennett

Synonyms. *Peach Red Suture*, Bennett, 1926; *Red Suture Virus*, Cation, 1932; *Peach Virus 5*, J. Johnson's classification.

The Virus and its Transmission. *Prunus Virus 4* is not apparently sap-inoculable, but it can be transmitted by budding. The insect vector is not known and there is no information on hosts for this virus other than the peach.

Disease Caused by *Prunus Virus 4*

Rosaceæ

Prunus persica (L.) Stokes. Peach. *Red suture disease*. The foliage symptoms on peach trees affected with red suture show best at ripening time, and the entire tree usually presents a yellowish green or bronzed appearance. Cation (16) describes the symptoms as follows: the tips of the leaves, particularly those that are twisted slightly downwards, are lighter in colour than the normal. The smaller leaves of a cluster are also often of a lighter colour. In advanced cases there is a distinct curving downwards and inwards with a twisting of the petiole on an occasional leaf, which throws the leaf parallel to the stem. The curving and twisting of the leaves differ for each variety of peach tree. Terminal growth is usually shorter than normal. Leaf clusters often sprout from most of the buds on a branch, and an unusual number of short shoots arise along the main branches towards the base. These

shoots have shortened internodes, spaces between the leaves or buds and are of small diameter, but are not long and wiry as in peach yellows (*Prunus Virus 1*). Such shoots, together with the abnormal number of leaf clusters sprouting on the main limbs near the centre of the tree, give the characteristic symptom described as "feathering" or "fuzziness," and this is not unlike a condition sometimes found in little-peach (*Prunus Virus 1 A*).

The fruit symptoms of red suture are most apparent at the time of picking and they are characteristic of the disease. Affected fruits ripen prematurely, usually several days early, and one side, usually the suture or crease side, softens first. The peach itself may have a bumpy or rough contour, with heavier ridges usually running lengthwise with the suture, or the ridge of the suture may be abnormally raised. The bumps on the fruit may at times be almost warty. Diseased peaches usually show an abnormal deep red to purple blush over the exposed side, and the colour is deepest on the apices or peaks of the ridges. The side of the peach which ripens first is abnormally soft and watery and is sometimes of a water-soaked appearance. The entire fruit when squeezed firmly in the hand is easily crushed under pressure which will not cause a normal peach to collapse.

Geographical Distribution. Red suture was first detected in Michigan, U.S.A., in 1911, and it is apparently confined to that State. It seems, however, to be gaining ground as the number of affected trees marked on 2,646 farms in 1931 was 1,434 as against seven on 1,841 farms in 1930.

Control. The control of red suture lies at the moment in the eradication of the diseased trees. All marginal cases should be removed and an effort should be made to plant trees only on the best peach soil and to keep trees in normal vigour to facilitate diagnosis of the disease (16). The virus yields to the same heat treatment that cures trees affected with *Prunus Viruses 1* and *1 A*.

PRUNUS VIRUS 5. Hutchins

Synonyms. *Peach Mosaic Virus*, Hutchins, 1938; *Peach Virus 6*, J. Johnson's classification.

The Virus and its Transmission. The virus is transmissible by grafting or budding, but not by sap-inoculation. Most of the stone-fruit trees are susceptible to a mosaic disease, but there is not sufficient evidence to show whether more than one virus is involved. In the present account, the mosaic diseases of the

various stone-fruit trees are dealt with under the heading of *Prunus Virus 5*, but the reader should realise that further research may necessitate an alteration in this arrangement.

The insect vector is thought to be *Anuraphis padi* (*Aphis pruni*), the plum aphid (see p. 509), though successful transmissions have so far only been made with this insect and the virus on plum trees. Atanasoff (4A) reports the following cross transmissions :

- (1) Apricot mosaic to plum.
- (2) Cherry mosaic to plum.
- (3) Cherry mosaic to peach.
- (4) Peach mosaic to peach.
- (5) Plum mosaic to cherry.
- (6) Plum mosaic to peach.
- (7) Plum mosaic to plum.

Diseases Caused by *Prunus Virus 5*

Rosaceæ

Prunus persica (L.) Stokes. The peach. *Peach mosaic*. The following account of peach mosaic as observed in the U.S.A. was kindly prepared for the writer by Dr. Lee M. Hutchins. The outstanding characters of the disease are as follows :

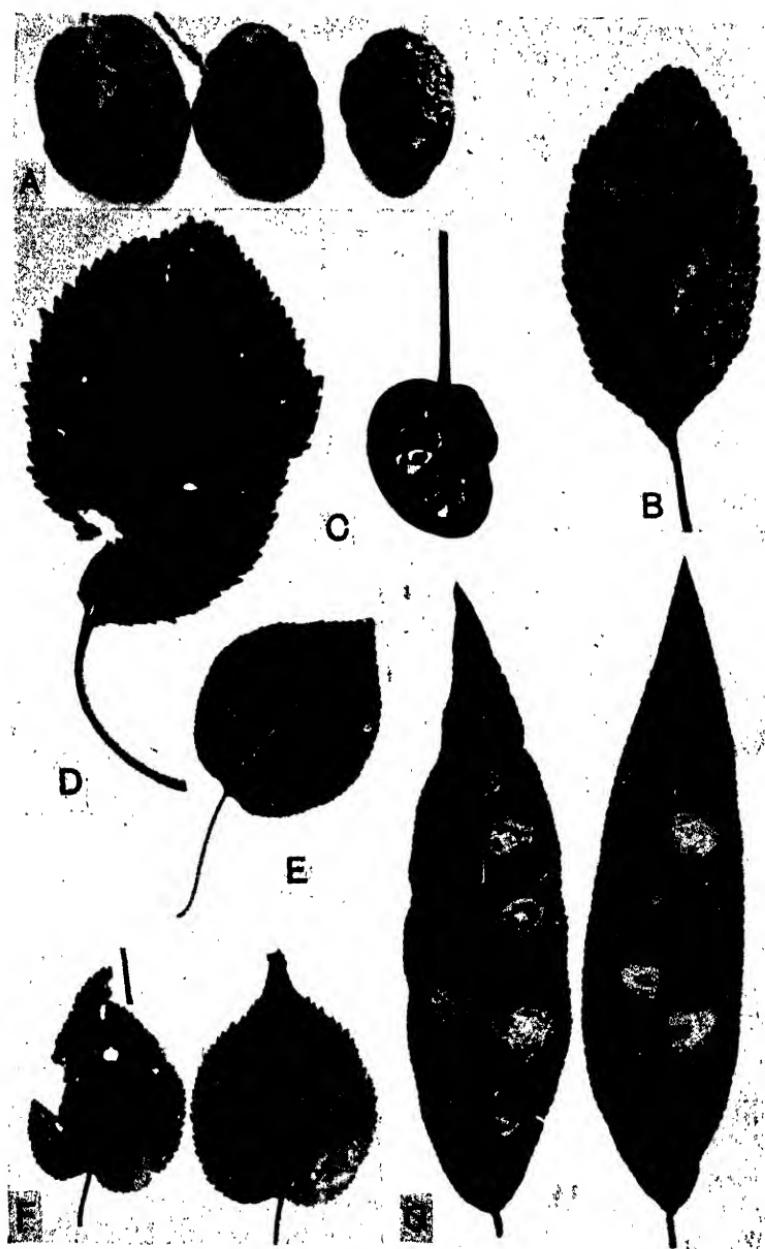
Tree. Marked dwarfing ; profuse branching, especially from twig terminals ; short internodes in spring, especially in the first inch or more of twig growth.

Flowers. No mosaic characters have been reported in peach flowers with the following exception : in blooms of large-flowered varieties in which the petals turn pink or red, Hutchins has observed in Texas that the disease may cause a conspicuous breaking of colour patterns similar to that which develops in mosaic-diseased tulips, wallflowers and violas (see pp. 411, 10, 55 respectively). This character was not discernible in the small-flowered varieties, such as Elberta.

Leaves. Conspicuous yellow mottling of the foliage in early spring is a constant symptom of peach mosaic in all varieties and under all conditions observed. This character disappears to a considerable extent by midsummer. Mosaic leaves are apt to be crinkly. The tender foliage is especially subject to injury from wind whipping, and this is often accompanied by the excision of small areas of leaf tissue (see Fig. 25, G).

Fruit. Smaller than normal, irregular in shape and, as a rule, bumpy along the suture, tip prominent. These characters are noticeably conspicuous in the Elberta and the J. H. Hale varieties.

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Mosaic fruit is generally unsaleable because of the unattractive appearance, small size and poor quality (see Fig. 24, B).

Seasonal Response. For the same variety, mosaic trees are a few days later than normal trees in coming into flower and leaf, and the fruit is apt to be a few days later than normal fruit in ripening.

Virulence. Under favourable conditions for its spread in commercial peach districts, peach mosaic is equally as dangerous as peach yellows, little-peach and phony disease (*Prunus Viruses 1, 1 A and 8*).

Prunus armeniaca L. Apricot. *Apricot mosaic* (see Fig. 25, F). The most common symptom of apricot mosaic is a faint mottling of the leaves which are sprinkled with lighter green spots, blotches and occasionally rings. The lighter green areas are not all sharply defined, but run out gradually into the normal background of the leaf. Here and there, more pronounced chlorotic spots, stripes or rings may develop in the lighter green blotches. Occasionally the light green blotches run along some of the main nerves or they may cover the area on both sides of the nerve, leaving narrow stripes of normal green along the nerves. The light green blotches may be numerous and small, or, on the other hand, there may be one or two larger blotches on each leaf. Only a small percentage of the leaves, even of the most severely infected trees, show symptoms of the disease. Atanasoff describes another form of mottling on leaves of the wild apricot and of one or two trees of the cultivated apricot. Here, very pronounced yellowish-green blotches and purple rings develop, followed by unequal growth of the leaves which gives rise to splitting of the leaf blade.

The most reliable symptom of apricot mosaic is to be found in the appearance of the fruit stones. Without exception, the stones of affected fruits show pronounced white rings and blotches. These white rings and blotches stand out very distinctly on the brown stones especially if the latter are wet. The fruits of affected

FIG. 25. *Prunus Virus 5* (peach mosaic virus).

- A. Fruits of mosaic-infected plum (plum pox).
- B. Leaf of mosaic-infected plum.
- C. Fruit of mosaic-infected cherry.
- D. Leaf of mosaic-infected cherry.
- E. Leaf of mosaic-infected *Prunus malaheb*.
- F. Leaves of mosaic-infected apricot.
- G. Leaves of mosaic-infected peach.

(After Atanasoff.)

apricot trees also show fairly definite signs of mosaic. The first symptoms are visible at ripening time and the fruits show very slight depressions and elevations on the surface. There are also blotches which are slightly elevated and of a lighter yellowish-green colour than the rest of the fruit (4A).

Prunus communis Fritsch. Almond. The symptoms on mosaic almond trees are very similar to those on peach and apricot. The exact type of mosaic which may be either in the form of rings or mottling, depends a good deal on the variety concerned. Infected almond fruits are similarly bumpy and irregularly developed.

Prunus domestica. Plum. *Plum pox*. The most common form of mosaic mottling on the plum, as shown on the Kustendil variety in Bulgaria, consists of a series of light green or yellowish-green blotches scattered in a fairly uniform manner over the leaf (Figs. 25, A and B). The blotches may, however, vary in appearance, sometimes they have the form of a streak running along the veins or they curl in a ribbon-like manner. Occasionally they develop into rings with a centre of normal green. Ring-like blotches are indeed characteristic of mosaic on stone-fruit trees as a whole.

Atanasoff (4A) describes several other types of mosaic symptoms on plums. In one of these, small irregular yellowish-green spots appear on the leaves, increase in number and size and finally unite to form large blotches which cover most of the leaf surface. In another type the veins are picked out in green on a chlorotic background, or, conversely, the veins appear chlorotic on a green background. On certain varieties the leaf mottling is very pronounced and resembles the variegations found in some ornamental plants. Towards the middle of the summer, the leaf blotches take on a pink or purple colour, finally becoming necrotic and dying out.

A third type of mottling on a variety of plum resembling Reine Claude consists of large lemon-yellow blotches with narrow ribbons of normal green tissue between. These ribbons of green tissue become slightly raised, like blisters, causing corrugation and distortion of the leaves. Plum trees bearing egg-shaped yellow plums when affected with mosaic sometimes show abnormal leaves. These leaves are not curled, but have some parts more expanded than others.

The fruits of affected plum trees, especially of the Kustendil plum, show pronounced and characteristic symptoms. The first sign is the development of olive-green watery spots under the

waxy bloom. Beneath these spots the pulp of the fruit is discoloured and watery, and as the fruit ripens the spots turn from olive-green to rusty-brown and purple. As a rule affected fruits ripen prematurely and the spots gradually sink in, forming small depressions or pock marks, usually at the top end of the fruits. It was these pock marks which suggested the name first applied to plum mosaic by Atanasoff, i.e., "plum pox."

Prunus avium L. Sweet cherry. *Buckskin of cherry* (see Figs. 25, C and D). There are two main types of leaf symptom associated with mosaic of the cherry. The first is of the mottling ringspot type and is the more common of the two and the second consists of streaks along the base of the mid-rib or side veins of the leaves.

The mosaic mottling of cherry leaves consists on some varieties of groups of blotches which may cover the whole or a part only of the leaf surface. Occasionally on such leaves may be seen several fairly distinct rings of lighter green tissue. The number of rings in each blotch varies from one to three or more. Usually at the centre of the blotch there is a small brown or white spot, not more than 1 or 2 mm. in diameter, then follows a ring of normal green tissue, and finally a ring of light green. Eventually the central spot of the blotch falls out giving the leaf a shot-hole effect. The mottling first becomes apparent when the leaves reach their full development in late spring and disappear during the height of the summer, reappearing towards the end of August.

The other type of symptom, described by Rawlins and Horne (75A) as the "buckskin" disease, consists, so far as the leaves are concerned, usually of a single streak along the base of the mid-rib. At first the streak is of a yellowish-green colour, later turning purple. According to Atanasoff (4A) this purple streak disappears in the summer and does not reappear, but Rawlins and Horne state that during early autumn the leaves on diseased trees show a peculiar reddish-purple coloration along the base of the mid-rib which extends out along the larger veins. This discrepancy can probably be explained by the fact that different varieties of cherry were under observation.

On the fruit the symptoms are quite characteristic and develop two or three weeks after blossoming. Small necrotic spots form in the green flesh, increase and unite to form a network of brown tissues. As the necrosis increases the fruit becomes brown from the tip down and falls prematurely. Some varieties of cherries show numerous but shallow and more or less uniform depressions

on the surface. In other varieties the depressions are deeper and reach down to the stone. In general, affected fruits are more or less conical in shape, have short pedicels and shrivel or fall before ripening (4A, 75A).

Geographical Distribution. The mosaic disease of stone-fruits or symptoms suggestive of it have been recorded from the following countries : Australia, South Africa, Germany ; in North America : Illinois, Kentucky, California, Minnesota, Texas, Colorado ; England, Holland, Czecho-slovakia, and Bulgaria.

Control. The three chief methods of combating plant virus diseases in general are also applicable for the control of mosaic of stone-fruit. These are "roguing" or eradication of affected trees, spraying to keep down the insect vector and the use of immune or resistant varieties. Atanasoff (4A) recommends that orchards should be carefully examined for signs of mosaic from four to six weeks after the development of the foliage. This examination should be repeated one or two months later, and all infected trees should be marked and removed as soon as possible. The inspection of orchards and nurseries and the marking of infected trees should be done while the trees are still rapidly growing and the foliage is still fresh. On the whole, symptoms of mosaic on all stone-fruit trees are more marked during the early summer, and it is therefore advisable to carry out the inspection before the summer is well advanced.

So far only the plum aphid, *Anuraphis padi* (see p. 509), has been shown to be the vector of the virus. This insect can be controlled by summer spraying with a contact insecticide such as nicotine or derris and by the use of egg-killing winter washes.

There is a certain amount of evidence that some varieties of plum and apricot are resistant to mosaic, and cherry trees grafted on to Malaheb (*Prunus malaheb* L.) stock seldom become infected with the virus. On the other hand, Atanasoff (4A) states that *Prunus malaheb* has been observed in Bulgaria showing distinct symptoms of mosaic (see Fig. 25, E).

According to Kunkel peach mosaic is not affected by the heat treatment which cures the other virus diseases of *Prunus*.

Finally, emphasis must be laid on the necessity for using perfectly healthy scions and stocks for propagation purposes.

PRUNUS VIRUS 6. Thomas and Hildebrand

The Virus and its Transmission. This virus is thought to be distinct from the other *Prunus* viruses, but nothing is known of

its properties or its natural mode of transmission and the distinction is made solely on a symptom basis. The virus has been transmitted to healthy trees by grafting and budding. It was first described in 1936 (84A).

Disease Caused by *Prunus* Virus 6

Rosaceæ

Prunus sp.. The prune. The affected leaves are reduced in size and distinctly narrowed in proportion to length. Serration and pubescence are suppressed and there is considerable rugosity and mottling. The rugosity is more marked near the mid-rib than toward the margin of the leaf. The leaf blade is somewhat thickened. Leaf margins are frequently so irregular in outline as to resemble, under casual observation, the effect of a biting insect. The surface of the severely affected leaf presents a somewhat glazed aspect. All the leaves on a given shoot are affected, those at the base somewhat more so than those at the tip. The shoot may grow for several inches in a season or only a fraction of an inch. Internodes are shortened in varying degrees. An unusual feature of the disease is the development of a shoot that remains perfectly normal in appearance throughout the season, in the midst of buds that produce only severely affected foliage. Diseased trees may blossom profusely, but only an occasional fruit matures on the parts that exhibit foliage symptoms. Pistils are aborted in many of the blossoms and petals are somewhat narrowed and irregular in shape. The time of blossoming and of maturity of fruit seems not to be influenced by the disease, and the quality of the few fruits that reach maturity seems normal.

Host Range. The virus seems to be confined to the plum and prune; attempts to infect the peach and the cherry have proved unsuccessful.

Geographical Distribution. At present the only record of the disease is from Niagara County, New York, U.S.A.

PYRUS VIRUS 1. Baur

Synonyms. *Pyrus Variegation Virus*, Atanasoff, 1935; *Infectious Chlorosis*, Baur, 1907, and Hertsch, 1930.

The Virus and its Transmission. There is no information concerning the virus which causes the variegation in mountain ash. The disease seems to be of the same nature as the "infectious variegation" of *Abutilon* spp., and Baur has shown that it can be transmitted by grafting (6).

Disease Caused by Pyrus Virus 1**Rosaceæ**

Pyrus aucuparia. Mountain ash. *Variegation*. The leaves of infected trees have at first yellow tips which later become white. In cases of severe infection the leaves do not show well-defined yellow zones, but become mottled with yellow spots. On some leaves the yellow tissues are limited to the tips. In other cases there is seen a clearing of the veins or alternatively a yellow band about 2 mm. wide may run along the main veins. The chlorotic tissues gradually become white and finally brown (4).

PYRUS VIRUS 2. Bradford and Joley

Synonym. *Apple Mosaic Virus*.

The Virus and its Transmission. Very little is known concerning this virus, it is apparently only transmissible by grafts or budding and not by sap-inoculation. The insect vector is not known, but one or more species of aphides may be suspected.

Diseases Caused by Pyrus Virus 2**Rosaceæ**

Pyrus malus L. The apple. *Apple mosaic*. The situation regarding the virus diseases of the apple is rather obscure. There seems little doubt that the apple tree is susceptible to infection with a mosaic virus, but it is not clear whether or not this is the same virus as that causing mosaic of peaches and plums.

An infectious variegation of the apple has been described by Bradford and Joley (11A) in which the chief symptoms consist of small spots on the leaves, some yellow and some creamy-white and irregular in outline. This disease is transmissible by grafting.

An apparent virus disease of apples in Kentucky in which the symptoms are mostly of the "ringspot" type has been briefly described (84B), and it is possible that this disease is the same as that described by Bradford and Joley.

A somewhat similar disease of apples has been investigated in Bulgaria (18A, 18B). Mosaic mottling in the form of pale green polygonal spots is most conspicuous on crab apples. On grafts the disease commonly assumes the form of chlorosis, accompanied by scorching of the leaf blade and in many cases by complete desiccation. At the same time necrosis sets in at the root tips and involves the phloem of the tap root and stem. Several varieties of three-year-old grafted apple trees have been observed with mosaic

symptoms. The fruit of all the affected varieties was shed prematurely. In the Grey French Pippin variety, a deformation of the corolla of the blossom was observed, similar to that in mosaic-attacked specimens of *Rosa gallica*. Slight deformation of the fruit occurs occasionally in mosaic-diseased apples such as Large Kassel Pippin and Winter Citron. The writer, also, has observed symptoms suggestive of mosaic on apple trees in this country.

Pyrus communis L. The pear. *Mosaic*. The following pear varieties have been observed by Christoff to be affected by a mosaic similar to that described on the apple, Enniszeika, Pastor, Edra Kasna, Hardenpont and other Butter types, Bonne Luise d'Avranches and Williams Bon Chrétien. The mottling on the leaves of pears is not of a very accentuated character, and on bearing trees a more or less pronounced chlorosis is often the only outward sign of disease. A characteristic of the disease is the formation of superficial, ill-defined spots on the pedicels; at first pale, these spots later turn blackish-brown in colour, and this is followed by the shedding of the young fruits. The fruits which remain on the tree develop dark green scattered or confluent spots mostly on one side and these are either superficial or involve the formation of scars.

Pyrus Cydonia. The quince. *Mosaic*. On one-year-old quince grafts the symptoms are similar to those described for the pear, but on bearing trees they are more variable. Deformation of the fruit is a marked feature on the Portuguese and Bereckzi varieties and in many cases the leaves show contortions and malformations of different kinds, accompanied by chlorosis and scorching. A further symptom in Portuguese quinces consists in a multiplication of the petals from five to as many as ten in a single flower. The fruits of the Portuguese and Bereckzi varieties may be deeply furrowed and sometimes deformed and grown together. The damage to the fruits is the most conspicuous and uniform symptom of quince mosaic; mottling or chlorosis of the leaves is relatively slight and may be altogether absent.

Host Range. According to Christoff (18A) the virus of apple mosaic is transmissible from apple to pear and quince. He also states (18B) that the virus from rose hips is transmissible to pears and apples, from plums, quinces and pears to apples and from apples to rose hips and pears.

Geographical Distribution. Infectious chlorosis or mosaic of apples has been recorded from Australia, the United States of

America in Michigan and Kentucky, Bulgaria and the British Isles.

Control. It is important to eradicate infected wild material from the neighbourhood of orchards and to avoid the use of scions from infected trees. Apple planting should be prohibited in heavily infested nurseries and routine spraying against aphides and other sucking insects should be carried out. Until the actual insect vector is identified, however, control measures can only be of a general nature.

ROSA VIRUS 1. White

Synonym. *Rose Mosaic Virus.*

The Virus and its Transmission. The virus is transmissible by grafts or buds, but not by sap-inoculation. The insect vector is not known. Diseased cuttings have been shown to remain infective after one hour's immersion in water at 45° C. and after fifteen minutes' immersion in 1 per cent potassium permanganate (66).

Diseases Caused by Rosa Virus 1

Rosaceæ

Rosa spp. Rose. *Rose mosaic* (see Fig. 24, C). The symptoms of this disease vary according to the variety of rose attacked. On hybrid tea roses the symptoms are as follows: the plants are dwarfed, the degree of dwarfing depending on the variety, severity of infection and environmental conditions. The dwarfing is expressed in all parts of the plant including the roots. The buds on infected plants are often bleached, imperfect and on short stems.

On seriously infected plants of the variety Madame Butterfly, the blooms are almost white instead of showing the normal light pink petals, tinted with gold at the base. The leaves are variously distorted with the mid-rib frequently bent and twisted. The leaflets show distinct chlorotic areas, especially along the mid-rib, which cause the leaflets to pucker and ruffle; occasionally the chlorosis is confined to one side of the leaf and this gives rise to distortion owing to the unequal growth of the two halves. Clearing of the veins is frequently pronounced. As a rule all the leaflets on a leaf show symptoms, but occasionally single leaflets occur, which are normal in appearance. According to White the variety of rose known as the Talisman is little affected by the

mosaic virus and diseased plants continue to produce good blooms (90).

Rosa manetti. There are two types of symptoms on this species, one consists of numerous, minute chlorotic areas, distributed over the entire leaflet or concentrated towards the tip or periphery of the leaflet. The other type is a more general chlorosis giving a typical mosaic-like mottle, and this type of symptom is more characteristic of glasshouse conditions while the former occurs on field-grown plants.

Rosa odorata. This plant has not been found naturally infected, but when infected by grafting from mosaic plants of Madame Butterfly, it showed a very marked distortion and dwarfing of the lower leaves, the stems in particular being mottled. Another type of symptom also develops on the older leaves, particularly on the upper surface of the leaflets. This consists of an irregular band of yellowish-green between the mid-rib and the periphery of the leaflet. Frequently the irregular leaf pattern is lost and the leaf will be blotched in definite areas, sometimes near the periphery and sometimes near the base. These symptoms develop only on mature leaves, and there is no dwarfing or malformation of the leaflets.

Rosa multiflora. The lower portions of the stems of this species bear dwarfed and malformed leaves but no other leaf symptoms. The stems themselves in these areas are mottled, but less so than in the foregoing species (89).

Host Range. The various cultivated roses including ramblers and other species of *Rosa* used as stocks all appear to be susceptible. McWhorter (60) suggests that two wild plants are hosts for the virus in Oregon and may serve as a source of infection, these are the wild wood rose (*Rosa gymnocarpa*) and the thimble berry (*Rubus parviflorus*). It has also been stated that the disease occurs in Pernetianas and hybrid Pernetianas (64).

As suggested already, there is some evidence of a connection between this virus and that causing mosaic of plum, and other stone-fruits, but this has not yet been proved.

Geographical Distribution. Rose mosaic appears to be widely distributed in the United States of America, in Oregon, Michigan, Kentucky and other States. The disease has been recorded in England on climbing roses and Manetti stocks, but does not appear to be common. It is also present in Bulgaria.

Control. Certain species of rose, such as *Rosa manetti* and others, much used for understocks, are very susceptible to infection

with the mosaic virus and care should therefore be taken that all stocks used are virus free. Spread of the disease may be kept down by careful roguing of infected plants.

ROSA VIRUS 2. Brierley

Synonym. *Rose Yellow Mosaic Virus*, Brierley, 1935.

The Virus and its Transmission. The virus is transmissible only by grafting and the insect vector is not known.

Disease caused by Rosa Virus 2

Rosaceæ

Rosa spp. Rose. A yellow variety of mosaic differing from that caused by *Rosa Virus 1* has been briefly described (18). It produces a chlorosis in affected roses of a lighter and brighter yellow than the ordinary rose mosaic. The disease has been observed on Hybrid Tea, Hybrid Perpetual and Hybrid Rugosa types of roses. It occurs in Ontario, New York, Maryland and Virginia.

ROSA VIRUS 3. Grieve

Synonym. *Rose Wilt or Dieback Virus*, Grieve, 1931.

The Virus and its Transmission. *Rosa Virus 3*, rose wilt virus, appears to be sap-inoculable and this property differentiates it from the two foregoing rose mosaic viruses which can only be transmitted by grafting. It is also filterable through a Seitz filter, but there is no information on other properties or insect vectors of this virus (31).

A virus disease of roses which seems similar to that caused by *Rosa Virus 3* has been recently described in Italy (29). In this case also the virus is sap-inoculable and the insect vector is said to be a species of *aphis* of the genus *Macrosiphum*.

Disease Caused by Rosa Virus 3

Rosaceæ

Rosa spp. Rose. *Rose wilt or dieback*. The first noticeable symptom is a peculiar recurved appearance of the leaves on young shoots, the leaflets of which sometimes also seem to be crowded together on the petiole and are very brittle. Defoliation begins at the tips of the stems of affected plants and works downwards, the leaves sometimes turning pale green or yellowish before dropping. About a day later the tips of the young stems begin to discolour and die back for a distance of 1 to 2 inches. The

next symptom is the development of a characteristic translucent yellowish-green appearance on the young stem, the base turning brownish-black within a few hours. Young leaf buds in the basal browned area remain green for some time. Gradually the whole stem becomes discoloured and dies back, the developing leaf buds turning brown at the tips and rotting away. In many cases the plant sends up one or more watershoots after the stems have died back, but they soon become discoloured. Temporary recovery may occur, but the plants wither in the end (31) (see Fig. 24, D).

Geographical Distribution. So far the only record of the occurrence of rose wilt is from Australia where it is said to be very prevalent among roses in Victoria and other parts of Australia. The disease generally occurs in epidemic waves, causing heavy damage for a season, subsequently losing virulence for a time and then regaining it.

ROSA VIRUS 4. Brierley

Synonym. *Rose Streak Virus*, Brierley, 1935.

The Virus and its Transmission. The virus of rose streak is not apparently sap-inoculable and has been transmitted by tissue union only. According to Brierley (12) this virus is distinct from both rose mosaic viruses and from that of rose wilt.

Disease Caused by Rosa Virus 4

Rosaceæ

Rosa spp. Rose. *Streak disease.* Brownish or reddish patterns of rings and vein-banding develop on the leaves of infected rose trees whilst the stems show ring patterns. On experimental infection by budding, certain hybrid tea roses such as Madame Butterfly, develop necrotic areas about the inserted bud. Commonly the stem is girdled at the site of the bud, the distal parts die and the leaves wither but persist. Frequently, nearly black secondary lesions with sharply defined margins, not depressed, appear on young lateral branches below the inserted bud (12).

Host Range. The streak disease has so far been recorded only on roses and has been found occurring naturally upon the following varieties and types of roses : the understocks *Multiflora* and *Odorata*, on different tea and hybrid tea roses, on hybrid Perpetual, hybrid *rugosa*, hybrid *wichuriana*, hybrid *multiflora*, *polyantha*, hybrid Bengal, China and Noisette classes.

Geographical Distribution. So far *Rosa Virus 4* has been

recorded only from the United States of America, where it occurs in Virginia, Columbia, Maryland and New York State.

PHASEOLUS VIRUS 1. Pierce

Synonyms. *Bean Mosaic Virus*, Stewart and Reddick, 1917; *Navy Bean Mosaic Virus*, Barss, 1921; *Azuki Bean Mosaic Virus*, Matsumoto, 1922; *Bean Mosaic Virus*, Fajardo, 1930; *Common Bean Mosaic Virus*, Pierce, 1934.

The Virus

Resistance to Chemicals. The resistance of *Phaseolus Virus 1* to alcohol is low, it is inactivated at strengths between 25 to 50 per cent. It is also inactivated by treatment for thirty minutes with a 1 : 500 solution of 37 per cent formaldehyde (27, 69).

Thermal Death-point. Inactivation occurs after ten minutes' exposure to a temperature of 56° to 58° C.

Dilution End-point. The dilution end-point is 1 : 1,000.

Resistance to Ageing. The longevity *in vitro* lies between twenty-four to thirty-two hours at laboratory temperatures.

Transmission. *Phaseolus Virus 1* is sap-transmissible, but only with difficulty. The addition of a small quantity of abrasive such as quartz sand or carborundum powder increases the number of successful transfers by this method (72). That this virus is transmitted through the seed was first proved in 1919 by Reddick and Stewart (77), and the phenomenon has since been observed by other investigators. Seed transmission, however, is irregular, and varies from 30 to 50 per cent; not all the seeds in one pod are necessarily infected. Plants infected during the growing season transmit the virus to the seed in a much lower percentage than those that have been diseased throughout the growing season. In the case of most infections that occur after flowering, the virus does not reach the seed (65). The early pods of seed-infected plants transmit the virus to a higher percentage of their seeds than do the late-formed pods (88). According to Reddick (76) *Phaseolus Virus 1* is also transmitted to healthy bean plants through the agency of the pollen of infected plants.

The insect vectors of *Phaseolus Virus 1* seem to be unusually numerous. No less than eleven species of aphides are stated to be capable of transmitting this virus (94). If this is the case, then the aphides are probably mechanical vectors only and other species are likely to be capable of transmission, the necessary

qualification being ability to feed upon the bean plant. It seems, however, essential for the potential vector to be an aphid. The following aphides are said to transmit *Phaseolus Virus 1*: *Aphis gossypii*, *A. medicaginis*, *A. rumicis*, *A. spiraecola*, *Brevicoryne brassicae*, *Hyalopterus atriplicis*, *Macrosiphum ambrosiae*, *Macrosiphum (Illinoia) pisi*, *M. gei (solanifolii)*, *Rhopalosiphum pseudobrassicae*, *Myzus persicae*.

Of these insects the most important are *Aphis rumicis*, *M. gei (solanifolii)* and *M. pisi*.

Further information on most of these aphides will be found in Chapter VIII.

Differential Hosts

Pierce (69) shows that certain varieties of bean (*Phaseolus vulgaris*) are extremely susceptible to *Phaseolus Virus 1*. When the primary leaves of the variety Refugee Green are inoculated symptoms first appear as a slight chlorosis of the first trifoliolate leaf, which tends to curl, but, ordinarily, shows no mottling. The leaflets of the second trifoliolate leaf become somewhat elongated, with the lamina curled downwards. Mottling occurs in various patterns, but, commonly, the background is of a somewhat lighter green than normal, with dark green areas interspersed on the light green field. The following varieties of beans appear to be resistant to infection: Robust, Great Northern, U.S. No. 5 Refugee, Idaho Refugee and Wisconsin Refugee. *Phaseolus Virus 1* is not transmissible to the sweet pea or garden pea.

Diseases Caused by *Phaseolus Virus 1*

Leguminosæ

Phaseolus vulgaris. French bean, Snap bean. The disease produced by *Phaseolus Virus 1* in the French bean is known as "common bean mosaic," and symptoms vary to a great extent according to the variety of bean affected, time of infection and environmental conditions. The following description applies to the disease as it occurs on the variety Stringless Green Pod Refugee (39). The leaf symptoms may be divided into two groups: *first*, early symptoms or those produced on leaves expanding at the time of infection; and *secondly*, typical symptoms or those produced on seed-infected plants and those developing subsequent to the early symptoms. The leaves that show the early symptoms are crinkled, chlorotic and stiff. The leaves droop and the petioles are much shortened. No definite downward rolling of the margins

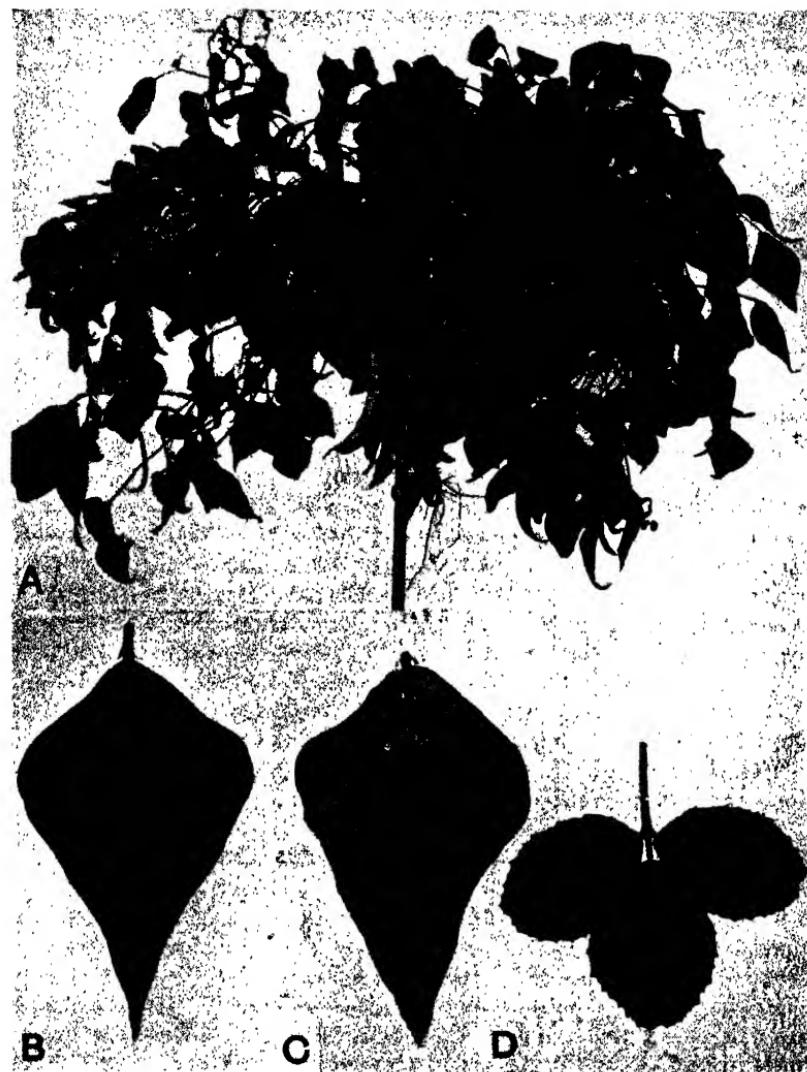


FIG. 26.

- A. Mature plant of bean (*Phaseolus vulgaris*), showing excessive branching of the vines due to infection with *Phaseolus Virus 1* (causing common bean mosaic).
- B. Leaf of bean (*P. vulgaris*), var. Stringless Green Refugee, showing type of mottling caused by *Phaseolus Virus 1*.
- C. Leaf of bean (*P. vulgaris*), var. Stringless Green Refugee, showing type of mottling caused by *Phaseolus Virus 2* (yellow bean mosaic virus).
- D. Symptoms caused by *Phaseolus Virus 2* on white sweet clover (*Melilotus alba*).

(A, after Harrison ; B and C, after Pierce ; D, after Zaumeyer and Wade.)

of the leaflets occurs nor is the characteristic mosaic mottling present. These early symptoms are not invariably shown by mosaic-infected plants (see Fig. 26, B).

On simple leaves showing the secondary symptoms there may be a general chlorosis of the leaf blade or a definite pattern of light green and dark green areas. The light green areas are usually along the margin of the leaf. On the compound leaves the typical or secondary symptoms vary considerably. A distinct downward rolling of the leaf margin usually occurs on the leaflets of the first few compound leaves formed on seed-infected plants and on the first few leaves produced after the leaves with early symptoms. Associated with the cupping is a distinct mosaic mottling composed of light green and dark green areas, the light green areas being more pronounced at the leaf margins. A characteristic symptom of mosaic on the first compound leaf and also the succeeding ones is the appearance of dark green blistered areas in the laminae. This blistering is generally contemporary with the marginal curling of the leaflet. All these leaf symptoms tend to become progressively less as the season advances. In addition, affected leaves are characterised by a shortening of the petioles, a proliferation of the vines, a general stunting of the plant and deformation of pods and flowers (see Fig. 26, A).

Pierce (69) has studied the symptom expression of common bean mosaic on twenty-four varieties of French beans and divides them into three classes according to their resistance to the virus. Class I. consists of susceptible varieties on which pronounced symptoms are produced. Class II. may be defined as the tolerant class. These varieties are infected as readily by inoculation as those in Class I., but do not ordinarily develop marked symptoms. The virus, however, may be recovered readily from inoculated plants even when no symptoms are visible. Class III. comprises those varieties of beans which are immune. They develop no symptoms on inoculation and the virus cannot be recovered from inoculated plants. Pierce (69) gives a list of eight susceptible, twelve tolerant and four immune varieties of beans.

A characteristic symptom of common bean mosaic under field conditions is the downward curling of the leaves, and this serves to differentiate the disease from yellow bean mosaic (see p. 161).

It will be seen, therefore, that the symptoms induced by *Phaseolus Virus I* (common bean mosaic) upon *P. vulgaris* are very variable, and depend on the variety, the age of the plant and

the environmental conditions. Affected leaves show, in general, various degrees of mottling and chlorosis, downward cupping of the laminæ, which gives an arched appearance to the leaflet, puckering and blistering. Mosaic-infected leaves may be smaller than healthy ones and very much contorted. On the less susceptible varieties a ruffling or crinkling of the leaves is characteristic of the disease, and this may be accompanied by a general chlorosis of the leaf with pronounced venation (95).

Histopathology. Sections through the chlorotic areas of mosaic-affected beans reveal a degeneration of the chloroplasts. Tangential sections through the petioles of mosaic bean plants are suitable for demonstrating destructive changes in the chloroplasts. In such sections, small islands of chlorotic cells, surrounded by normal-appearing cells, may frequently be observed. The affected cells are pale yellow and structural changes in the plastids are common. The stroma becomes flattened and larger in diameter, and the chloroplast eventually collapses into a coherent mass of viscous, pale yellow or colourless material. Another type of chloroplast destruction consists in the development of small vacuolated areas in the stroma. Within the vacuolated areas, small hyaline bodies or granules are always present (65). Symptoms tend to be masked at temperatures above 30° C. and below 15° C. (89).

Host Range of *Phaseolus Virus 1*. The following leguminous plants are susceptible to infection with the virus of common bean mosaic: *Phaseolus acutifolium*, var. *latifolius*; *P. lunatus* L.; *P. calcaratus* Roxb.; *P. aureus* Roxb.; *Vicia faba* L.; *V. lathyroides* L.; *Lespedeza striata* Hook and Arn.; and possibly *Melilotus alba* Desr. *Phaseolus lunatus*, var. *Macrocarpus* and *P. limensis* are very difficult to infect and should probably be regarded as resistant. As already stated, the virus does not appear to be transmissible to the sweet pea (*L. odoratus*) and garden pea (*Pisum sativum*) (69).

Geographical Distribution. Common mosaic of bean, now known to be caused by *Phaseolus Virus 1*, was first recognised by Iwanowski in Russia as early as 1899, and by Clinton in Connecticut, U.S.A., in 1908. Symptoms of mosaic have been observed in young seedlings of beans from twenty-two different countries, and the disease has now been reported from forty-two States in the U.S.A. It is clear, therefore, that *Phaseolus Virus 1* is of very wide distribution and is probably co-extensive with the host (65).

PHASEOLUS VIRUS 2. Pierce

Synonyms. *Yellow Bean Mosaic Virus*, Pierce, 1934; probably *White Sweet Clover Mosaic Virus*, Zaumeyer and Wade, 1935; *Pea Mosaic Virus 2* of Zaumeyer and Wade, 1935.

The Virus

The physical properties of this virus are very similar to those of *Phaseolus Virus 1*.

Resistance to Chemicals. The virus is inactivated by 50 per cent alcohol and by 37 per cent formaldehyde at a dilution of 1 : 1,000.

Thermal Death-point. Exposure for ten minutes to temperatures between 56° to 58° C. inactivates the virus.

Dilution End-point. The dilution end-point is about 1 : 1,000.

Resistance to Ageing. Infectivity is lost after twenty-four to thirty-two hours at laboratory temperatures.

Transmission. The virus is sap-transmissible and infection is rendered easier by the addition of carborundum powder to the inoculum. It is not transmitted by the seed, an important difference from *Phaseolus Virus 1*.

The insect vectors are the aphides *Macrosiphum (Illinoia) pisi*, the pea aphid, and *M. gei*, the pink and green potato aphid (see pp. 582 & 584), and possibly other species.

Differential Hosts

Phaseolus Virus 2 can be differentiated from *Phaseolus Virus 1* by the more severe disease it produces in *P. vulgaris* and by the fact that it is transmissible to certain varieties of the garden pea (*Pisum sativum*).

Diseases Caused by Phaseolus Virus 2**Leguminosæ**

Phaseolus vulgaris. The French or snap bean. *Yellow bean mosaic* (see Fig. 26, C). The symptoms of yellow mosaic on the bean, var. Refugee Green, as described by Pierce (69), are as follows, the disease is much more virulent than the common bean mosaic. Each leaflet is not only curled downwards, but, in addition, is definitely *pointed downwards* from the place of attachment to the petiole. This is a simple method of distinguishing between common bean mosaic and yellow bean mosaic. The surfaces of the leaflets are slightly irregular, and small light yellow spots soon develop in the dark green background.

The yellowing gradually spreads over the entire surface, causing the leaflets to become more or less chlorotic. In these early stages the young growth has a tendency to become brittle. The first trifoliate leaflets do not remain curled downwards, but, as they enlarge, become slightly concave on their upper surfaces and take on a glossy appearance. On the third and fourth trifoliate leaves there is a very distinct mottling of yellow green and dark green areas which stand out in greater contrast to each other than is the case in the common bean mosaic. The downward curling usually associated with the latter is not a constant characteristic of yellow bean mosaic in the later stages of development. In plants affected with yellow bean mosaic, symptoms do not become masked in their later stages of growth, but the mottling becomes more pronounced as the season progresses. Plants become decidedly stunted and bushy because of a reduction in the length of the internodes and a proliferation of branches. Maturity is delayed and the production of pods greatly reduced. In certain varieties of beans necrosis of the young leaves is a symptom of yellow mosaic, while in others there is a tendency to malformation.

Pisum sativum. Garden pea. On certain varieties of garden peas *Phaseolus Virus 2* produces a typical mosaic pattern. This mosaic is somewhat milder than the disease caused by *Pisum Viruses 1 and 2*.

The first signs of this disease, as with that caused by *Pisum Virus 2*, consist of a faint mottling which later becomes more intense owing to the presence of numerous dark green areas, irregular in outline and occurring between the larger veins. Later a pronounced vein-clearing appears and immediately adjacent to the larger veins the dark green tissue often persists. The region between the veins remains green, but is of a lighter shade than in the normal plant. Especially along the periphery of the leaves, regions of yellow are often found. The leaves and stipules of the infected plant are smaller than normal, but there is only a slight waving and upward curling of the edges and wrinkling of the leaves. Infected plants are only slightly distorted, a point of difference from common pea mosaic (*Pisum Virus 2*), where the distortion is considerable, though the pods may be somewhat malformed and distorted and in some cases reduced in size.

Melilotus alba. White sweet clover. Symptoms first appear as small light yellow spots on the leaves. These spots may enlarge and coalesce with others, producing small light green blotches

interspersed with dark green areas. Frequently there is a clearing of the veins, with the dark green islands situated between them. Severe infection may cause slight dwarfing and ruffling of the leaves (see Fig. 26, D).

Host Range of Phaseolus Virus 2. The virus of yellow bean mosaic is transmissible to all the species given as susceptible to *Phaseolus Virus 1* with the exception of *Phaseolus lunatus* and *P. calcaratus*. In addition *Phaseolus Virus 2* will infect white sweet clover, *Melilotus alba* Desr.; soybean, *Soja max* Piper; white lupin, *Lupinus albus* L.; crimson clover, *Trifolium incarnatum* L.; alsike clover, *T. hybridum* L.; and black medick, *Medicago lupulina* L. This virus, unlike *Phaseolus Virus 1*, is transmissible to garden pea (*Pisum sativum*).

Geographical Distribution. There is little information on the occurrence of this disease outside the United States of America, where it seems to be widely distributed on white sweet clover (*Melilotus alba*).

PHASEOLUS VIRUS 3. Zaumeyer and Wade

Synonym. *Bean Mosaic Virus 3*, Zaumeyer and Wade, 1935.

The Virus and its Transmission. This virus was first described upon a hybrid plant of *P. vulgaris* by Zaumeyer and Wade (95), and was considered by them to be distinct from the two preceding bean viruses. There appears to be no information at present on the properties of the virus or its insect vector.

Diseases Caused by Phaseolus Virus 3

Phaseolus vulgaris. The French or snap bean. *Bean mosaic.*

The virus was first noticed causing a very mild chlorosis on the hybrid of Stringless Green Refugee × Wells Red Kidney. The symptoms were very faint and there was no malformation of the leaves and no stunting of the plant. On the variety Stringless Green Refugee the first symptoms produced by *Phaseolus Virus 3*, ten days after inoculation, are characterised by an extremely chlorotic and dwarfed condition of the trifoliolate leaves immediately above the inoculated ones. Later formed leaves may show a very marked clearing of the veins and veinlets bordered with normal green tissue. Later the remainder of the interveinal tissue turns yellow, resembling in general effect a calico pattern. The leaves of diseased plants are smaller than those of normal plants, but there is no wrinkling, curling or other malformation.

Control of Phaseolus Viruses 1 and 2. The problem of the control of bean mosaic may be approached from various aspects, these different methods include the use of mosaic-free seed, the roguing of mosaic-infected plants and the selection of mosaic-resistant or immune stocks from commercial lots of seed and, finally, the breeding of a mosaic-immune bean. Harrison (39) has found that so far as *Phaseolus Virus 1* is concerned, every sample of commercial seed tested carried the virus. He also found it impracticable to eliminate the virus from the seed, since any which survived the various sterilisation methods sufficiently to germinate were still liable to carry the virus. Roguing mosaic-infected plants is considered to be impracticable as a general rule because of the rapidity of spread of the infection. It might, however, be used in bean fields situated on exposed slopes where experience has shown that the spread of mosaic is less rapid.

The production of mosaic-resistant or immune varieties would be, of course, the best means of controlling the disease. It is considered that the chances of obtaining such varieties by selection from the field are not great, and that resistant varieties will have to be obtained by breeding. While this is likely to be a long task, it may be mentioned that the Wisconsin Agricultural Experiment Station, in co-operation with the Idaho Agricultural Experiment Station, has introduced two varieties, Wisconsin Refugee and Idaho Refugee, which are immune from bean mosaic. Further, it has been shown that certain mosaic-resistant varieties of French bean possess a different type or degree of resistance, and that it is possible to predict with a reasonable degree of accuracy the results that may be expected from crosses of these various resistant varieties with susceptible types (70).

The Idaho Agricultural Experiment Station has developed several strains of "Great Northern" field beans by selection which are immune from common bean mosaic (*Phaseolus Virus 1*). The U.S. No. 5 Refugee, developed by the United States Department of Agriculture, is also resistant to this virus.

Certain cultural practices may aid somewhat in the reduction of bean mosaic ; these consist in the selection of exposed situations and the isolation, so far as practicable, of the bean field from other leguminous crops such as clover (39).

SOJA VIRUS 1. Gardner and Kendrick

Synonyms. *Soybean Mosaic Virus*, Gardner and Kendrick, 1921 ; *Soybean Virus 1*, Pierce, 1985.

The Virus.

Thermal Death-point. Inactivation occurs after exposure for ten minutes to a temperature of 58° C.

Resistance to Ageing. The longevity *in vitro* of this virus is not more than three days (70).

Transmission. The virus is sap-inoculable, and it is also carried in the seed in a percentage of cases. The insect vector has not been determined.

Disease Caused by Soja Virus 1

Leguminosæ

Soja max. Soya bean. *Soybean Mosaic.* Affected plants are stunted and the petioles and internodes are somewhat shortened. The leaflets are stunted, greatly misshapen and puckered with dark green puffy areas along the veins. Between these puffy areas the leaf tissue is etiolated. Affected leaflets tend to be asymmetrical, twisted, and curled downwards about the margins. The pods on mosaic plants are stunted and flattened, less pubescent and more acutely curved than those on normal plants. Pods borne at the upper end are more severely affected. Diseased plants tend to remain green longer than normal plants (28).

Host Range. The virus seems to be confined in its range to the soybean. It is not transmissible to bean (*Phaseolus vulgaris*).

Geographical Distribution. *Soja Virus 1* is widely distributed in the U.S.A., and since it is seed-borne it probably occurs wherever the soybean is grown.

PISUM VIRUS 1. Osborn

Synonyms. *Pea Virus 1*, Pierce, 1935; *Enation Pea Mosaic Virus*, Osborn, 1935; *Pea Virus 1*, Stubbs, 1936.

The Virus

Thermal Death-point. The virus is inactivated by a temperature of about 56° C., no infection being obtained after exposure for ten minutes to 58° C. (71).

Dilution End-point. This is 1 : 8,000.

Resistance to Ageing. Inactivation of the virus occurs after three days' ageing at laboratory temperatures.

Transmission. The virus is sap-transmissible, but with difficulty, and the addition of carborundum powder to the inoculum increases the number of infections. There is no evidence of seed transmission.

The insect vectors according to Osborn (68) are the aphides

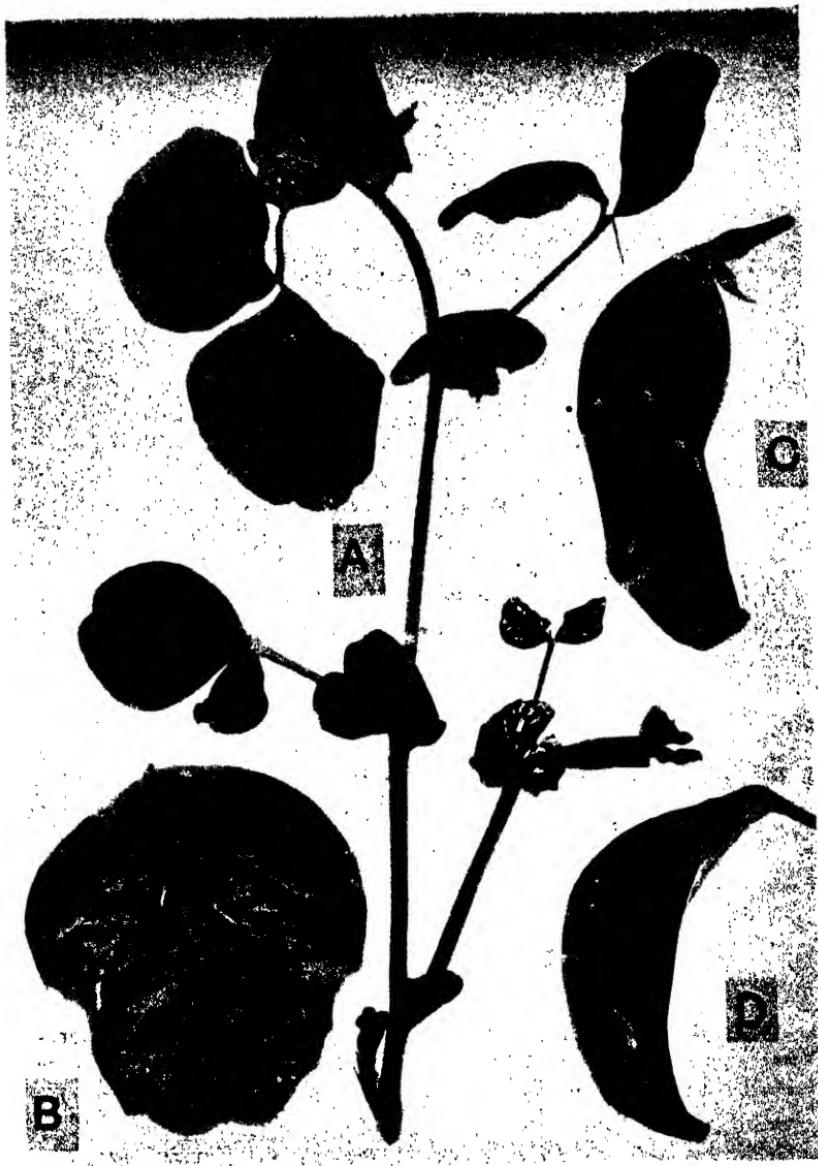


FIG. 27. *Pisum Virus 1* (enation pea mosaic virus).

- A. Infected pea seedling, var. Alderman, showing pronounced vein clearing, distortion of top foliage and transparent spots on secondary stem foliage.
- B. Lower surface of pea leaflet, showing the leaf enations characteristic of infection with this virus.
- C and D. Two pods showing deformation and enations.
(A and B, after Stubbs ; C and D, after Pierce.)

Macrosiphum gei, Kalt. and *M. pisi* (see pp. 532 & 534), but not *Aphis rumicis*.

There is a delay in the development of infective power within the aphid which varies from nine to forty-eight hours. Aphides which once acquire the virus may retain it for considerable periods of time, sometimes as long as twenty-nine days.

Differential Hosts

This virus is not transmissible to French bean (*Phaseolus vulgaris*), but it is inocutable to the American pea varieties, Perfection and Horal, which are resistant to *Pisum Virus 2*.

Diseases Caused by Pisum Virus 1

Leguminosæ

Pisum sativum. Green or garden pea. *Enation pea mosaic*. The symptoms produced by *Pisum Virus 1* on peas are usually very severe and consist of mottling, crinkling and savoying of the leaves and stipules. Yellowish spots develop on the leaves and later become white and somewhat transparent. On very susceptible varieties like Alderman necrotic spots appear, accompanied by proliferations on the under surfaces of the leaves. Since these enations are not found on leguminous plants affected with the other viruses they are considered to be of special diagnostic value (71). When the pea plant becomes infected prior to completion of pod development the pods become markedly distorted. The ovary wall assumes a rough, ridged, wrinkled condition and, as a result of these corrugations, is badly deformed and somewhat dwarfed. The ridges usually appear dark green in colour. Pods on a badly diseased vine may be so severely twisted as to be hardly recognisable. The seeds in diseased pods sometimes appear smaller and yellower than normal. Upon holding an opened mosaic pod to the light the zones responsible for the raised irregularities of the outer surface appear dark green in contrast to the green of the rest of the pod (80) (see Fig. 27).

Vicia faba. Broad bean. *Broad bean mosaic*. Diseased plants are characterised by a conspicuous mosaic pattern that appears on the leaves. The pattern takes the form of a spotting rather than a mottling, the spots being somewhat irregular in shape and variable in size. They tend to appear along the veins and in some plants expand into broad stripes. Symptoms of this disease have occasionally been observed five days after inoculation, but they

usually appear in from six to fourteen days. Stunting sometimes occurs in diseased plants, but is usually not severe (68).

Soja max. Soybean, var. Mid-west. On soybean, *Pisum Virus 1* produces a mottled dark and light green mosaic pattern.

The virus is also transmissible to sweet pea (*Lathyrus odoratus*) on which it produces a mosaic.

Geographical Distribution. *Pisum Virus 1* (enation pea mosaic virus) has been recorded chiefly from the United States of America, and it appears to be of some importance in California. Böning (11) describes a disease of the broad bean (*Vicia faba*) in Germany which may be due to this virus. There is no information on its distribution in the British Isles.

PISUM VIRUS 2 Doolittle and Jones

Synonyms. *Pea Virus 3*, Pierce ; *Pea Virus 1*, Zaumeyer and Wade ; *Pea Mosaic Virus*, Doolittle and Jones ; *Common Pea Mosaic Virus* ; *Pea Mosaic Virus*, Osborn, 1935 ; *Red Clover Mosaic Virus*, Zaumeyer and Wade, 1935 ; *Pea Mosaic Virus*, Chamberlain, 1936.

The Virus

Thermal Death-point. The virus is inactivated by ten minutes' exposure to temperatures of 60° to 64° C.

Dilution End-point. The virus loses infectivity at about 1 : 1,500 dilution.

Resistance to Ageing. The virus is still infective after ageing for forty-eight hours, but fails to infect after seventy-two hours at 22° C.

Transmission. The virus is sap-transmissible with an abrasive, and the insect vector is the aphid *Macrosiphum pisi*, and, according to Chamberlain (18) *Myzus persicae* and *Aphis rumicis* (see Chapter VIII).

In a paper just published, Osborn (*Phytopath.*, 1937) states that there is no delay in the development of infective power within the aphid vectors which are able to acquire, and transmit, the virus after five-minute feeding periods on diseased and healthy plants respectively.

The virus does not seem to be carried in the seed. Murphy and Pierce (*Phytopath.*, 1937) grew 4,268 seedlings from plants of Alderman and Dwarf Alderman pea severely affected with common pea mosaic without a single case of seed-transmitted

mosaic. It is therefore probable that the high percentage of seed transmissions reported by Dickson (23) were due to the use of a different virus.

Differential Hosts

Pisum Virus 2 is not transmissible to *Phaseolus vulgaris*. The American varieties of pea, Perfection and Horal, are resistant to this virus but susceptible to *Pisum Virus 1*.

Diseases Caused by Pisum Virus 2

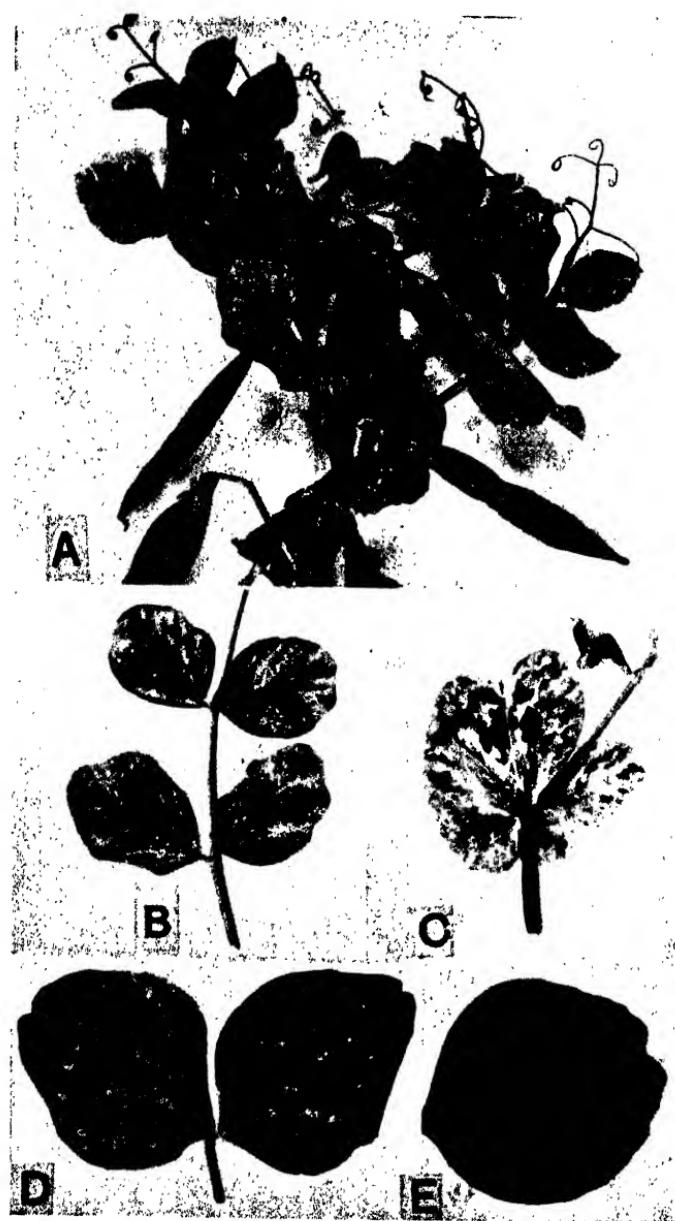
Leguminosæ

Pisum sativum L. Garden or green pea. *Common pea mosaic*. The symptoms produced by *Pisum Virus 2* vary considerably, depending upon the age of the plant, and upon the environmental conditions. The first symptom following inoculation to garden pea is a clearing of the veins in the leaves of the new growth. Later symptoms are characterised by chlorosis or severe yellowing of the leaves with numerous dark green areas dispersed over the leaflets (Fig. 28, B). General stunting of infected plants is a typical and characteristic symptom. The symptoms are usually most severe in the upper portions of the plant, and a general chlorosis may or may not be apparent. Certain varieties such as Alaska and Telephone tend to show general chlorosis, while other varieties such as Alderman, World's Record and Market Surprise tend to show distinct mottling (Fig. 28, A).

Lathyrus odoratus L. Sweet pea. On the sweet pea this virus produces pronounced mottling and chlorosis. Dark green areas are found interspersed in the abnormally yellow-green portions of the leaves and the flowers have "broken" colours (Fig. 29).

Vicia sativa L. Common vetch. Symptoms on common vetch are characterised by mottling and severe curling of the leaves.

Vicia faba. Broad bean. *Broad bean mosaic*. The writer has observed plants of *V. faba* infected with a mosaic disease thought to be due to the action of *Pisum Virus 2*. The leaves show a bright yellow and green mottle (see Fig. 29). The symptoms first become apparent when the pinnate leaves begin to show above the leaf base. Occasionally the affected leaves are abnormally narrow and elongated. The healthy areas along the mid-ribs and accessory veins of mottled leaves are sharply delimited from the pale diseased portions of the leaf. Histological changes in the tissue



become recognisable at the same time as the external symptoms. Primarily affected leaves remain with the palisade and spongy parenchyma undifferentiated and the diseased areas are thinner than normal (11).

Lupinus angustifolius. Lupins. *Sore shin disease*. The first visible symptoms are a slight stunting of the plant associated with the characteristic curling of the growing point to one side. Simultaneously a light brown streak appears on that side extending the whole length of the stem. Growth ceases with the appearance of the first symptoms. On cutting, the vascular system of infected plants shows a brown discolouration which is more extensive in the roots and growing points than in the main stem. The discoloured area subsequently extends until the whole stem becomes brown. The young leaves at first wilt and then turn black, while the older ones become tinged with purple, turn yellow and ultimately fall. The stem turns black, the roots decay, and, finally, the plant dies (18, 63). The leaf symptoms differ according to the variety of lupin affected. In the commercial varieties of yellow lupins (sweet and bitter) the leaf curls inwards, while the leaflets bend upwards towards one another in a claw-like manner and exhibit some mosaic symptoms with brown stripes and spots. In blue lupins the growing tips become limp, the lower leaves become violet or brownish and the whole plant soon dies. In the white variety the leaves are much curled with the margins outwards, small yellowish spots and stripes develop while the lowest leaves hang limply downwards. On young affected plants the flowers frequently fail to develop; on older plants, infected later, the young pods are black or spotted with black and droop instead of standing up obliquely to the stem (81).

Trifolium pratense L. Red clover. *Melilotus officinalis*. Yellow sweet clover. On these two species of clover *Pisum Virus 2* produces a mosaic mottling which, on red clover, is very similar

FIG. 28. *Pisum Virus 2* (common pea mosaic virus).

- A. Infected pea plant, var. Alderman, showing the characteristic mottling.
- B. Mosaic mottling on leaflets of Alderman pea.
- C. *Pisum Virus 2A* (marble strain).
- D. *Pisum Virus 2B* (speckle strain).
- E. *Pisum Virus 2C* (mild strain).

The symptoms of the three strains are all on Alderman pea.
(A, after Zaumeyer; B, after Pierce; C-E, after Stubbs.)

to that produced by *Phaseolus Virus 2* on the same plant and consists of a definite yellow mottling (see Fig. 29).

Host Range of *Pisum Virus 2*. The host range of this virus has recently been studied by Murphy and Pierce (*Phytopath.*, 1987). Their studies included a total of 2,424 plants, representing thirty-two families, sixty genera, and ninety-four species. No infection with *Pisum Virus 2* was obtained in any plant family other than the family Leguminosae. Three hundred and thirty leguminous plants of nine genera, twenty-eight species, and thirty-two horticultural varieties were found susceptible to *Pisum Virus 2*.

Of the susceptible host plants the more important are the following: chick pea, *Cicer arietinum* L.; *Desmodium canadense* (L.) D.C.; sweet pea, *Lathyrus odoratus* L.; grass pea, *L. sativus*; blue lupin, *Lupinus angustifolius*; white lupin, *L. albus*; *L. hartwegii* Lindl.; *L. nanus* Dougl.; *L. densiflorus* Benth.; spotted burr clover, *Medicago arabica* Huds.; toothed burr clover, *M. hispida* Gaertn.; white sweet clover, *Melilotus alba* Desr.; annual yellow sweet clover, *M. indica* All.; yellow sweet clover, *M. officinalis* Lam.; tepary bean, *Phaseolus acutifolius*, var. *latifolius* Freem.; *Trifolium procumbens* L.; crimson clover, *T. incarnatum* L.; *T. reflexum* L.; *T. dubium* Sibth.; *T. agarium* L.; *T. carolinianum* Michx.; persian clover, *T. suaveolens*; alsike, *T. hybridum*; red clover, *T. pratense* L.; cluster clover, *T. glomeratum* L.; broad bean, *Vicia faba* L.; and common vetch, *V. sativa* L.

Strains of *Pisum Virus 2*

Three mosaic diseases of the garden pea (*Pisum sativum*) which seem to be caused by slightly different strains of *Pisum Virus 2* have recently been described in America (84). They are differentiated from each other only on a symptom basis, but all three are differentiated from *Pisum Virus 2* by the fact that they are not transmissible to red clover, *Trifolium pratense*.

Pisum Virus 2 A. Common Pea Mosaic Virus (Marble Strain).

Stubbs

The first symptom to appear is vein-clearing in the newest leaves and stipules produced during the six- to eight-day incubation period. The next set of leaflets and stipules produced are much reduced in size, somewhat distorted, and always more or less chlorotic. Nearly complete chlorosis of the most recently



FIG. 29. *Pisum Virus 2* (common pea mosaic virus).

(Top) Infected broad bean, *Vicia faba*.

(Bottom) (left) Infected red clover, *Trifolium pratense*; (right) infected sweet pea, *Lathyrus odoratus*; note breaking in the flower colour.

(Bottom, left and right, after Zaumeyer and Wade.)

formed foliage is usually to be observed at this stage of disease development. Foliage produced by further growth bears the "marble" type of mottle pattern, which consists of rather large chlorotic areas bounded by light green or normal green tissue. Much of the chlorotic tissue is almost colourless. Other symptoms include leaf-drop on the lower part of infected plants and yellowish-brown stem discolorations at the nodes. Such stem discolouration appears first at the point of leaf petiole attachment. Later the discoloured area usually completely encircles the stem and then spreads along the internode (see Fig. 28, C).

Pisum Virus 2 B. Common Pea Mosaic Virus (Speckle Strain.)
Stubbs.

Disease symptoms caused by this strain differ from those caused by *Strain 2 A* in type of mottle and degree of expression of the other symptoms noted. The general course of disease development is the same in both cases. The mottle pattern produced by *Strain 2 B* consists of small irregular shapes of dark green tissue bounded by extensive yellowish-green areas. There are a few almost colourless chlorotic areas. There is very little distortion or reduction in size of leaflets and stipules; a small amount of cortical discolouration is usually restricted to the node areas. Leaf-drop is occasionally present (see Fig. 28, D).

Pisum Virus 2 C. Common Pea Mosaic Virus (Mild Strain.)
Stubbs.

First signs of infection with this strain commence about eight days after inoculation and consist of a very slight vein-clearing. The next two or three sets of leaflets and stipules produced exhibit a very mild mottle. The foliage does not become chlorotic enough to make the mottle pattern conspicuous. Growth of plants is not retarded and no distortion of foliage occurs (see Fig. 28, E).

Geographical Distribution. *Pisum Virus 2* seems to have a wide distribution; it is frequently observed in the British Isles and in Europe generally, and it is apparently widespread over the United States of America. It is also present in New Zealand, where it causes a widespread disease of blue lupins as well as peas.

Control. In addition to endeavours to produce resistant varieties of peas, some control of the viruses affecting this plant may be obtained by measures against the insect vector, *M. pisi*. The following spray is recommended: ground derris root containing 8·2 per cent of rotenone and 18 per cent of total extractives,

used at a dilution to obtain 0·0115 per cent of rotenone (3 lb. per 100 gallons of water), and one of two spreaders and wetting agents, a sulphated butylated diphenol at a dilution of 1 : 600 of a 40 per cent aqueous solution or a sodium oleyl sulphate at a dilution of 1 : 1,000 (14).

TRIFOLIUM VIRUS 1. Pierce

Synonyms. *White Clover Virus 1*, Pierce, 1935; probably one constituent of the *White Clover Mosaic* of Zaumeyer and Wade, 1935.

The Virus

Resistance to Chemicals. According to Zaumeyer and Wade this virus is not inactivated by 1 : 100 hydrochloric acid, but it is destroyed by 75 per cent. alcohol and 1 : 200 formaldehyde solution.

Thermal Death-point. The virus is inactivated by exposure to a temperature of 58° C.

Dilution End-point. Infectivity is lost at dilutions of 1 : 2,000 or over.

Resistance to Ageing. The virus retains its infectivity for six to seven days at laboratory temperatures according to Pierce (71), but Zaumeyer and Wade (95) consider that it is inactivated after ageing for thirty-two hours.

Transmission. The virus is sap-inoculable, but the question of seed-transmission is not yet settled. Zaumeyer and Wade suggest that the virus may be so carried on occasion. The insect vectors are thought to be *Aphis rumicis* and *Macrosiphum (Illinoia) pisi* (see pp. 524 & 584) since Merkel (61) transmitted a virus of white clover (*Trifolium repens*) by means of these two species.

Differential Host

According to Zaumeyer and Wade, *Trifolium Virus 1* produces local necrotic lesions on the inoculated leaves of French bean (*Phaseolus vulgaris*) (see Fig. 30, B). These lesions may appear in thirty-six to forty-eight hours after inoculation, as small, somewhat circular light green areas. Later the lesions become brownish-red, often accompanied by a slight clearing in the centre which is usually surrounded by a dark ring of tissue, while outside this is a region of lighter brown to red tissue (95). Pierce, however, was unable to produce definite local lesions on *Phaseolus*;

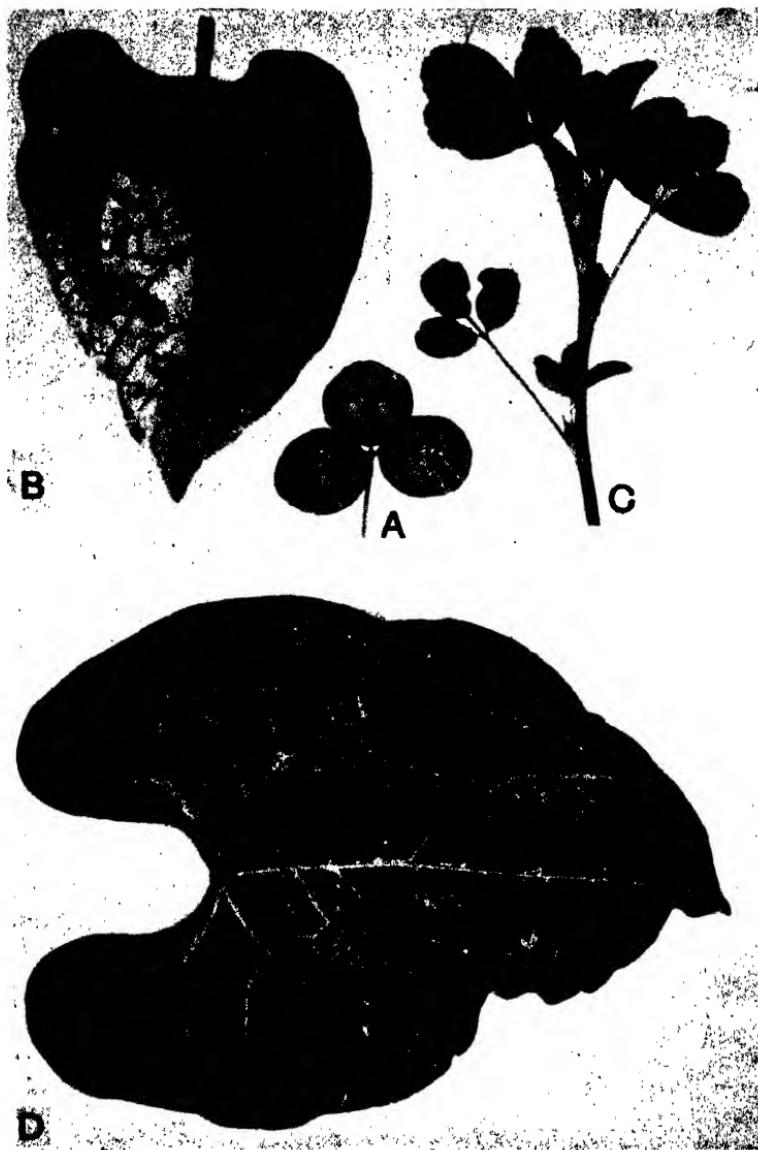


FIG. 30.

- A. *Trifolium Virus 1* (white clover mosaic virus) on white clover.
- B. Local lesions caused by *Trifolium Virus 1* on bean (*Phaseolus vulgaris*), var. Robust.
- C. Mosaic of lucerne (alfalfa) caused by *Medicago Virus 2*.
- D. Local lesions caused by *Medicago Virus 2* on bean (*Phaseolus vulgaris*), var. Stringless Green Refugee.

(After Zaumeyer and Wade.)

usually a systemic mild mosaic only developed, but in some instances a slight necrosis was evident on the inoculated leaves.

Diseases Caused by *Trifolium Virus 1*

Leguminosæ

Trifolium repens L. White clover. *White clover mosaic* (see Fig. 30, A). The disease first manifests itself on the leaves as a slight mottle of light and dark green areas. The darker green regions are usually found adjacent to the main vein and extending outward along the laterals, becoming lighter towards the margin of the leaflet. Occasionally the greater portion of the leaflet may be quite chlorotic with only the basal portion streaked with green. Islands of dark green areas are frequently found in other portions of the leaf. Infected leaves are usually not distorted, but occasionally there is slight puckering and arching along the main vein. The symptoms may be readily masked under certain environmental conditions (95). According to Pierce (71), what is presumably the same virus causes particularly severe symptoms on red clover (*Trifolium pratense* L.) and yellow sweet clover (*Melilotus officinalis* L.). Distinct mottling is accompanied by crinkling of the leaves, and in some cases a slight spot necrosis is evident on red clover leaves. Yellow sweet clover plants are definitely dwarfed, crinkled and mottled when infected with this virus.

Phaseolus vulgaris. French bean. Systemic infection develops in ten to eleven days after inoculation, but it should be mentioned, however, that according to Zaumeyer and Wade there may be some doubt if this systemic infection is due to one virus alone. The symptoms consist of blotches of light green or yellow areas on the trifoliolate leaves, irregular in outline. They are not bounded by the veins, but extend throughout any portion of the lamina. These blotched areas may coalesce with similar areas, in some cases covering three-quarters of the leaf surface, leaving only small islands of normal green tissue. The infected leaves may show a slight ruffling, but their size is not reduced nor is the leaf malformed as is often found in the case of common bean mosaic (*Phaseolus Virus 1*). Infected plants are not stunted, and under greenhouse conditions the symptoms may be readily masked. Under field conditions the disease might be mistaken for mild infections of common bean mosaic (95).

Vicia faba. Broad bean. Some necrosis is caused in broad beans infected with *Trifolium Virus 1*; the plants are not killed,

but show a distinct mottling, together with some malformation and necrotic spots in the leaves.

Pisum sativum. Garden pea. Pierce (71) states that *Trifolium Virus 1* causes a necrotic disease in peas which under winter glasshouse conditions becomes systemic and fatal. In some instances a severe systemic mosaic may develop.

Geographical Distribution. There is little exact information on the distribution of *Trifolium Virus 1*. It appears to be fairly widespread in the United States of America, and is probably present in Europe as well.

Note. The *White Clover Virus 1* of Pierce, and *White Clover Mosaic Virus* of Zaumeyer and Wade are evidently the same virus or strains of the same. However, the local lesion phase on beans, described by Zaumeyer and Wade, was not obtained by Pierce. Since Zaumeyer and Wade reported two thermal death-points for their virus, the question arises as to whether or not they were working with a virus mixture, possibly *Trifolium Virus 1* and *Medicago Virus 2*.

MEDICAGO VIRUS 1. Weimer

Synonyms. *Alfalfa Virus 1*, Weimer: *Common Alfalfa Mosaic Virus*, Weimer, 1934.

The Virus and its Transmission. The virus is not sap-inoculable and there is, in consequence, no information on its properties. The insect vector is the aphis, *Macrosiphum (Iliinoia) pisi* Kalt (see p. 584).

Differential Hosts

This virus unlike *Medicago Virus 2* is non-transmissible to beans and peas.

Disease Caused by Medicago Virus 1

Leguminosæ

Medicago sativa L. Alfalfa, Lucerne. *Common alfalfa or lucerne mosaic*. The first evidence of the disease in a leaf is the appearance of one or more small, more or less circular, greenish-yellow spots. These areas frequently consist of a yellowish band of tissue, $\frac{1}{2}$ to 1 mm. in width, surrounding an island of apparently normal colour, $\frac{1}{2}$ to 2 mm. in diameter. In some cases there are two or more concentric rings of green surrounding narrow bands of chlorotic tissue.

The chlorosis gradually spreads until eventually the rings are more or less obscured. There may be only one spot on a leaf, but more frequently there are several. These spots often coalesce, involving considerable areas of the leaf. The affected tissue is greenish-yellow at first, but gradually becomes lighter, as more chlorophyll is broken down, until it is distinctly yellow, and in extreme cases, especially where the leaf remains on the plant for a long time, almost white in colour. The chlorosis may spread in all directions from the central ring so that a considerable amount of the tissue may eventually become involved. Although the rings may lie between or on the veins, the yellow colour characteristically runs parallel to the veins and frequently involves them. Besides being chlorotic, affected leaves may be reduced to one-third their normal size, crinkled, and more or less deformed, although this happens only in severe cases, usually in the spring before the first cutting when the disease is most conspicuous. In some leaves there may be little evidence of ring formation, although there may be considerable mottling. Severely affected leaf tissue often is considerably thickened and brittle, breaking easily when bent. There appears to be some dwarfing of the affected stems when the disease is severe, but, for the most part, there is no visible effect on the growth of the plant. No necrotic lesions have ever been seen on the stems. Only the lower leaves of a plant or all of them, including those not fully expanded, may be affected. Diseased spots do not fall out, but remain intact throughout the life of the leaf. The disease does not cause premature defoliation (87).

Geographical Distribution. *Medicago Virus 1* has been recorded from California, and is probably present in various parts of the United States of America.

MEDICAGO VIRUS 2. Pierce, Zaumeyer and Wade

Synonyms. *Alfalfa Virus 1*, Pierce; *Alfalfa Mosaic Virus*, Zaumeyer and Wade.

The Virus

Resistance to Chemicals. According to Pierce this virus is inactivated by treatment with 75 per cent alcohol, but Zaumeyer and Wade find it still infectious after such treatment. It is inactivated by thirty minutes' treatment with nitric acid at 1 : 200, by hydrochloric acid at 1 : 100 and by 1 : 100 concentration of 87 per cent formaldehyde.

Thermal Death-point. Infectivity is destroyed by exposure for ten minutes to temperatures between 62° and 64° C.

Dilution End-point. Infectivity is lost at a dilution of about 1 : 2,000

Resistance to Ageing. The longevity *in vitro* of the virus, according to Pierce, is from seven to nine days, and, according to Zaumeyer and Wade, three to four days.

Transmission. The virus is sap-transmissible, and the insect vector is the aphid *Macrosiphum (Illinoia) pisi*, the pea aphid (see p. 584). There is no evidence of seed transmission. Pierce found that thirty-four seedlings grown from seed obtained from Manchu soybean plants, artificially infected with the virus, were all healthy (69).

Differential Hosts

Phaseolus vulgaris. French bean. When inoculated to French beans, *Medicago Virus 2* produces small, reddish-brown, local lesions two days after inoculation. Some of the spots are surrounded by an irregular ring of tiny lesions of the same colour. The larger type of lesion varies from 0·5 to 2·0 mm. in diameter. With age, the irregular ring of small lesions coalesces with the larger one and a clearing occurs in the centre. The lesions differ from those produced by *Trifolium Virus 1* in that they are smaller in size and their edges are more regular. The virus does not become systemic (95) (see Fig. 80, D).

Unlike the majority of mosaic viruses affecting leguminous plants, *Medicago Virus 2* is transmissible to garden petunia, *Nicotiana tabacum* and *N. glutinosa*.

On broad bean, *Vicia faba*, the virus produces a very pronounced vascular necrosis which finally kills the plant.

Diseases Caused by Medicago Virus 2

Medicago sativa L. Alfalfa, Lucerne. *Alfalfa Mosaic.* There is very little information on the type of mosaic symptoms caused on lucerne by this virus. Pierce (69) obtained it originally from a strain of Turkestan lucerne. The affected plants were decidedly dwarfed and the leaves distinctly mottled and crinkled (see Fig. 80, C).

Host Range. In addition to the plants included under "differential hosts," Pierce (69) gives the following host list: hyacinth bean, adzuki bean, mung bean, rice bean, common and

Turkestan alfalfa or lucerne, white sweet clover, crimson clover, red clover, white clover, "Perfection" pea, spring vetch, Manchu and Mikado soybeans.

Strains of *Medicago* Virus 2

Zaumeyer (98) has recently differentiated three strains of this virus. The symptoms produced by these viruses on pea are similar to one another except in intensity. Strains 2 A and 2 B produce a leaf mottling, while Strain 2 C produces a leaf spotting in addition to mottling.

Geographical Distribution. The only record of the type virus appears to be from Wisconsin, U.S.A.

MEDICAGO VIRUS 3. Weimer, 1936

Synonym. *Alfalfa or Lucerne Dwarf Disease Virus*, Weimer, 1931.

The Virus and its Transmission. The virus is not sap-inoculable but it can be transmitted by grafting. The insect vectors are not known, but the disease has been observed to spread in the field. The insertion of pieces of tissue from diseased plants into wounds in healthy plants fails to produce infection except when the yellow xylem of the root or crown is used. Any method of grafting in which diseased and healthy root tissue forms a union gives a high percentage of transmission. One hundred per cent transmission can be obtained by grafting a piece of diseased root on to the lower end of the taproot of the healthy plant. There is no information available on the properties of this virus (88).

Disease Caused by *Medicago* Virus 3

Leguminosæ

Medicago sativa. Alfalfa or lucerne. *Dwarf or little-leaf disease*.

So far as is known at present this virus is confined to alfalfa or lucerne. The earliest stages of the dwarf disease cannot be detected by the symptoms above ground, since the disease is already well advanced in the root before it becomes evident in the top. The first signs of the disease in the tops are a shortening of the stems and a slight reduction in the size of the leaves. Blossoming is often retarded or inhibited. Usually no chlorosis or other colour change occurs in the leaves or stems until the last few stems die. In the final stages of the disease only one or at most a very few stems are produced, and these reach an ultimate

height of only a few inches. These stems remain upright and for the most part turgid until death ensues. Not infrequently the leaves of diseased plants appear to be of a darker green than those of healthy plants. The leaves of affected plants are not mottled, crinkled or deformed, although commonly they are somewhat rounded at their apices, resembling more closely the basal than the terminal leaves of healthy plants. The stems of diseased plants are reduced more or less uniformly in size.

Root Symptoms. The first evidence of the disease in the root is a small yellow streak in the wood, apparent only when the bark is removed. This streak, which varies in size, may occur anywhere in the root, especially in the upper foot of the taproot. As the disease develops the yellowing spreads until it eventually involves the entire circumference of the root. When the root is cut across, the yellow colour is found in the outermost part of the woody cylinder just beneath the bark. This discoloured tissue forms a definite ring or band which is narrow at first, but becomes wider as the disease develops, until at the time the plant dies the root is frequently discoloured throughout its entire diameter. The yellowing extends into the main divisions of the crown and into the base of the green stems, but does not penetrate very far into the latter (86).

Geographical Distribution. *Medicago Virus 3* has so far been reported only from southern California.

MEDICAGO VIRUS 4. Edwards

Synonym. *Lucerne Witch's Broom Virus*, Edwards, 1936.

The Virus and its Transmission. Nothing is known at present concerning the properties of the virus since it is not sap-transmissible. The insect vectors are not yet identified. Positive transmission has been obtained by means of grafting, using the side or veneer grafting method.

Disease Caused by Medicago Virus 4

Leguminosæ

Medicago sativa. Alfalfa, lucerne. *Witch's Broom*. The disease caused by this virus has been called Lucerne Witch's Broom by Edwards (25), and it is known variously by growers in New South Wales as "Spindle Shoot," "Mistletoe," "Kurrajong" or "Bunchy Top."

The most characteristic symptom of the disease is a marked

dwarfing of the plant. The leaves are considerably reduced in size and are typically more rounded than normal. They often exhibit marginal chlorosis and distinct puckering and distortion of the laminæ. The stems are very short and are usually much finer than normal and there is invariably a considerable increase in the number produced. Typically the diseased plants produce a thick, dense mass of shoots not exceeding a few inches in height and having an erect bunched appearance, the leaves being rounded and reduced to about one-third the normal size. The diseased plants differ in colour from the normal and may be either lighter or darker than healthy plants. In the early stages of infection a slight general chlorosis is often evident and the plants have a definite yellowish tinge, but later when proliferation has occurred the foliage is often very dark in colour and may have a marked purplish tinge. The majority of the diseased plants fail to flower, but occasionally blooms may be produced which are considerably smaller and paler in colour than the normal inflorescence. Occasionally also the floral parts are replaced by leafy structures. Seed is rarely produced, although slight seed production has been observed on several occasions. The failure of the diseased plant to flower normally and produce seed is one of the most striking features of the disease in areas which are climatically suitable for heavy seed production by normal plants. In the later stages of the disease the crown and upper root tissues are often severely rotted and the plants gradually die. An interesting feature of the disease is the masking of symptoms which may occur in the early spring growth. In this case the plants are normal in appearance except for the increase in the number of stems. After cutting back, however, these plants will again show the characteristic features of the disease in the subsequent growth (25).

Geographical Distribution. The virus causing the witch's broom disease of lucerne is widespread in New South Wales; it is also present in Queensland, Victoria and South Australia. What is most probably the same virus has been recorded in Utah and Idaho, U.S.A.

Differentiation and Identification of the Constituent Viruses in Complex Diseases of Leguminous Plants

Leguminous plants, particularly beans, peas and the various clovers, are liable to infection with several viruses simultaneously in a similar manner to the potato plant, but in a lesser degree. It will be useful, therefore, to indicate some possible methods by

which such disease complexes may be analysed. When combinations of *Phaseolus* Viruses 1 and 2 are suspected, inoculation to certain American varieties of French bean, such as Robust, Idaho Refugee and Wisconsin Refugee, will isolate *Phaseolus Virus 2*, since these varieties are immune from *Phaseolus Virus 1*. To isolate this latter virus it may be necessary to obtain it from the seed, since only *Phaseolus Virus 1* is seed-transmitted. In combinations of *Medicago* Viruses 1 and 2, the latter may be separated by the mere act of sap-inoculation to a susceptible host since *Medicago Virus 1* is not sap-inoculable.

Pisum Virus 1 may be separated from *Pisum Virus 2* by inoculating the complex to the American pea varieties, Perfection and Horal, which are susceptible only to *Pisum Virus 1* (enation pea mosaic virus). *Pisum Virus 2* can probably be isolated from the mixture of the two viruses by reason of its higher thermal death-point. This virus can also be obtained free from admixture with any of the three strains 2 A, B, and C by inoculation to *Trifolium pratense*, which is susceptible to the type virus but resistant to the three strains.

These methods of separation thus briefly outlined are only intended to indicate the lines which such separation technique should follow and the student should endeavour to supplement them by further inquiry into the differential properties of these viruses.

ROBINIA VIRUS 1. Hartley and Haasis

Synonym. *Robinia Brooming Disease Virus*, Hartley and Haasis, 1929.

The Virus and its Transmission. There is no information available on the insect vectors or properties of the virus, which has only been transmitted by grafting.

Disease Caused by Robinia Virus 1

Leguminosæ

Robinia pseudacacia. Black locust. *Brooming disease*. The chief symptom of this disease, which is thought to be due to virus infection, is the development of a characteristic witch's broom. There are proliferation and reduction of both shoots and leaves and the brooms are always erect or ascending. On a single tree, 7 inches in diameter, the crown of which was two-thirds broomed, "cushions" were found on the trunk. The typical cushion is 4 to 5 cm. in diameter and approximately 1 cm. thick. The

cushion bears groups of buds and tiny leaves on its surface and is regarded merely as an extreme form of broom. On older trees branches which bear brooms die back from the tips ; the brooms themselves are also short-lived. The disease is frequent on sprouts arising from the roots and stumps of trees that have been cut. In some cases the entire sprout is a witch's broom, making a low tiny-leaved plant. More often the brooms develop terminally or in axils of normal leaves near the top of the sprout. Among larger trees the disease is rare, but its frequent appearance on sprouts following cutting operations makes it seem possible that many of the older trees are infected without showing recognisable symptoms.

This disease has only been described from the United States of America, where it has been recorded in five States of the Union (40).

ARACHIS VIRUS* 1. Zimmermann

Synonyms. *Groundnut (Peanut) Rosette Disease Virus*, Storey and Bottomley, 1928 ; *Groundnut "Krauselkrankheit" Virus*, Zimmermann, 1907.

The Virus and its Transmission. Nothing is known of the properties of this virus. It is not carried in the seed or soil and is apparently not sap-inoculable. The insect vector is *Aphis laburni Kalt.* (*Aphis leguminosae* Theo.) (see p. 517).

Diseases Caused by Arachis Virus 1

Leguminosæ

Arachis hypogaea L. Groundnut, peanut. *Rosette disease.* This virus produces a striking modification in the peanut plant ; the whole plant may be little more than a close tuft of small curled leaves, forming a "cushion" of a few inches diameter, or its branches may be of some length, but bear terminally, similar tufts of small leaves. Accompanying this rosette form of injury is a yellowing, usually distributed over each of the young leaves, but occasionally confined on each leaf to irregular areas, separated by normal green tissue, producing a mosaic-like pattern.

The abnormalities bringing about this divergence from the normal plant form may be analysed into the following symptoms : a cessation of the growth of the axis, a reduction in the length of the petiole and in the size of the leaflets, and chlorosis, malformation and curling of the leaflets.

The first sign of infection is a faint indefinite mottling of the

youngest leaflets. The next leaf to open is predominantly of a pale yellow colour upon which the veins form a green network. This distinctive character is of diagnostic value for the rosette disease at this stage. Later-formed leaves bear progressively smaller leaflets, chlorotic (often uniformly yellow, without dark veins), curled and distorted. Elongation of the axis ceases after the appearance of the first chlorotic symptoms.

The occurrence of mosaic-like markings shows considerable variability, and may be absent from a typical rosetted plant. In some plants, however, the leaflets exhibit a characteristic and pronounced mosaic pattern ; this condition is usually accompanied by less severe stunting symptoms. The rosetted plant may flower, but few of the pegs make any growth and none bear seed. The only yield from diseased plants is the seed formed before the plant became diseased ; where infection has occurred early in the plant's growth, the crop is a total loss (83).

Host Range. According to Brooks (15) numerous other plants in the Gambia have been observed showing typical rosette symptoms. It is not known, however, whether attempts were made to infect healthy groundnuts from these plants. The following have been observed apparently infected : *Petunia*, *Vinca*, *Calliopsis*, *Calendula*, *Chrysophyllum cainito* and *Lagerstroemia*.

Geographical Distribution. The rosette disease of groundnuts has a wide distribution over tropical and sub-tropical Africa. It has been recorded from the Gambia, Senegal, Madagascar, Sierra Leone, Uganda and also from Java.

Control. Probably the best means of controlling the rosette disease will be the introduction of resistant varieties. In the Gambia some success has already been achieved in this direction by the production of the two varieties Philippine Pink and Philippine White, which show considerable resistance to the disease.

In some parts of Africa early sowing is recommended, but in others it is considered that the date of the first appearance of the disease depends mainly on seasonal factors, being little affected by the date of sowing. The following observations refer to the disease under conditions of growth in the Gambia (44), late plantings are more susceptible to rosette than early plantings ; wide spacing gives more disease than close planting ; the disease appears to be associated with periods of drought ; the outside plants appear to be more liable to infection than those in the middle of the plot ; the presence of weeds tends to inhibit the

spread of rosette. This last observation suggests that either soil moisture or atmospheric moisture around the plant is an important factor, since weeds tend to prevent evaporation from the soil and induce dew formation. This in its turn may possibly affect the habits of the insect vector.

Strains of Arachis Virus 1

There is some evidence of the existence either of more than one rosette virus or of several strains of the type virus. In the Gambia, Hayes (44) differentiates three distinct disease types which he designates "Chlorosis Rosette," "Green Rosette" and "Type No. 3." The first is characterised by yellow and pale green patches on a dark green background, the second by an abnormally dark green coloration of the leaves without chlorosis, and the third by a thickening of the stem and a reduction in the size of the leaves together with some thickening of the latter.

It is possible that the chlorosis rosette and the green rosette are due to the action of two distinct viruses. If plants infected with each type of disease are grafted together, symptoms of chlorosis rosette develop in the plant showing green rosette, while the green rosette disease appears in the chlorotic plants. This lack of cross immunity between the two suggests that they may be separate entities (15).

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CHAPTER III

Ficus Virus 1 ; Humulus Viruses 1-4 ; Santalum Viruses 1, 1A and 2 ; Vitis Virus 1 ; Apium Viruses 1-2 ; Vaccinium Virus 1 ; Dahlia Viruses 1, 2, 2A and 3 ; Callistephus Viruses 1 and 1A ; Lactuca Virus 1.

FICUS VIRUS 1. Condit and Horne

Synonym. *Fig Mosaic Virus*, Condit and Horne, 1933.

The Virus and its Transmission. The virus is not sap-inoculable, but can be transmitted by grafting. It is not seed-borne. The insect vectors have not been discovered, but it has been suggested in California that the fig scale insect, *Lepidosaphes fici* Sign., and the mite *Eriophyes fici* Ewing, might be concerned in the transmission of the virus (6).

Disease caused by Ficus Virus 1

Moraceæ

Ficus Carica L. Fig, var. White Ischia. *Mosaic*. In mosaic of the fig as observed in England (1), leaf symptoms of two types occur. Some leaves exhibit irregular yellowish-green blotches, often with paler coloured margins and up to $\frac{1}{2}$ inch or more across, distributed on the leaf blade with little relation to the veins, while other leaves show pale green spots or bands, usually associated with the larger veins. These latter spots often have narrow, reddish-brown margins. The coloured areas are quite superficial and are inconspicuous when the lower surface of the leaf is examined. The leaves are not noticeably deformed. An occasional fruit may show a few spot-like markings.

In California the disease seems more severe, as the leaves are distorted, the fruit is frequently spotted, and there may be premature fall of both leaves and fruit.

There is considerable difference in varietal reactions to the disease. In California one variety, an entire-leaf caprifig form of *Ficus palmata* Forsok., was considered to be immune, others were found to be somewhat resistant, and the White Ischia and Celeste varieties were stated to be among the more susceptible types.

Geographical Distribution. Fig mosaic seems to be widely

distributed ; it has been reported from several localities in the British Isles, from California, and from New South Wales.

Control. At the moment no control measures are known ; care must be taken, however, to see that stocks are never propagated from mosaic-affected trees.

HUMULUS VIRUS 1. Salmon

Synonyms. *Hop Mosaic Virus* : *Hop False Nettlehead Virus*, Duffield, 1925.

The Virus and its Transmission. The virus of hop mosaic is apparently not sap-inoculable, and there is no information on its manner of spread in the field. It is not considered to be carried in the seed or the soil. The virus can be transmitted by grafting. Nothing is known of the properties of the virus.

Disease caused by Humulus Virus 1

Cannabinaceæ

Humulus Lupulus L. The hop plant. *Hop mosaic*. The first symptom in an affected plant is the yellowish-green mottling of the leaves, which become brittle and curled, with recurved margins. The tip of the stem, or bine, is also somewhat brittle ; it is unable to climb and falls away from the string. All the affected vines with arrested growth are barren ; the diseased shoots may either remain green through the season or they may die off during the summer. The roots, when examined, are found to be partly dead. In cases of late infection the bine may reach the top of the string or pole and produce a varying amount of "hops," but the presence of the disease is shown by the curling and mottling of the leaves and by the fact that some of the hop-cones show curious and characteristic malformations. The disease is usually fatal to the hop plant, which dies in one or two years. So far as is known at present, the virus attacks only the hop plant. Certain varieties of hops, though infected with the mosaic virus, yet show no symptoms. These are known as "carriers" (see viruses affecting the potato), and are quite common amongst commercial varieties of hops. Certain male hops are also known to be carriers (24, 26).

Geographical Distribution. The virus of hop mosaic seems to be fairly widespread in Europe ; it is especially common in England. It has been recorded from Czechoslovakia, Germany and Poland, and it is thought to have been found in the United States of

America. Legislation to prevent the importation of mosaic-infected hops is in operation in Poland and Australia.

Control of Humulus Virus 1. Careful "roguing" of affected plants together with the immediate neighbours is an important measure. Cuttings should never be taken from infected plants, and, if possible, not from hop gardens where mosaic is known to be present. The use of mosaic resistant varieties, such as "Fuggles," is also to be recommended in gardens where the disease is prevalent. Until the natural method of spread of the hop mosaic virus is known it is difficult to devise other methods of control.

HUMULUS VIRUS 2. Duffield

Synonyms. *Hop Nettlehead Virus*: "Eelworm" Disease: possibly the same as the *Kräuselkrankheit* or *Curl Disease* of hops in Germany, Poland and Czechoslovakia.

The Virus and its Transmission. Nothing is known of the physical properties or natural means of spread of this virus. It is not sap-inoculable, but has been transmitted by grafting (28).

Disease caused by Humulus Virus 2

Cannabinaceæ

Humulus Lupulus L. The hop plant. *Nettlehead disease*. This disease chiefly affects the Fuggles variety. Diseased plants show continued weakness of growth, the hill producing a large number of weak shoots, somewhat resembling a cluster of nettles. The bines reach only a few feet up the pole or string, and their tips are unable to climb and hang away from the string. The leaf, which often shows an abnormally long terminal lobe, is curled upwards and inwards at the margin. Affected plants produce no cones or only a few worthless ones; the disease is rarely fatal (25).

Geographical Distribution. The disease has been known for about fifty years in England, but there do not appear to be any other authentic records. A somewhat similar disease of hops is known in Czechoslovakia, Germany and Poland.

HUMULUS VIRUS 3. Salmon and Ware

Synonym. *Hop Chlorotic Disease Virus*, Salmon and Ware, 1930.

The Virus and its Transmission. There is at present no information on the physical properties of this virus. It is

transmissible by budding and by grafting and is also sap-inoculable, but the insect vectors, if any, are not known. The virus appears to be seed-transmitted (29).

Diseases caused by *Humulus Virus 3*

Cannabinaceæ

Humulus Lupulus L. The hop plant. *Chlorotic disease.* In a plant which has already made growth and has started to climb some or all of the primary leaves exhibit pale yellow or greenish-yellow areas on the lamina. When the leaf is viewed against the light the colour of the affected parts is primrose-yellow. On an otherwise normal leaf the chlorosis appears most commonly near the extreme edge of the basal lobes of the cordate lamina, but in a leaf which is more diseased the yellow colour extends in a narrow band close beside the veins, sometimes widening to eliminate any intermediate strip of green, and so forming a completely yellow area between the veins. The abnormal coloration may cover only a small part of the lamina, or it may be intermixed with the ordinary green colour in an almost equal proportion; again, it may invade practically the whole lamina, leaving only the extreme tip of the lobes with healthy colour. The extent of attack seems to be determined at an early stage of development of the leaf, and instances have not been observed in which the chlorosis has spread in a fully expanded lamina. When the yellow colour occurs near the margin of the leaf, it is commonly associated with excessive serration or with complete absence of serration. Occasionally the serrations remain green and then turn upwards in the form of a fringe. Leaves having large chlorotic areas are commonly distorted, the green parts continuing growth and developing into domed or bulbous parts, and those affected with chlorosis remaining only partly expanded and restricting the even development of the entire lamina. Lack of growth at the chlorotic margin serves to increase the distortion, and parti-coloured leaves, closely down-curled, are a frequent, though not constant, feature of the disease.

Histopathology. Sections of chlorotic leaves show a great reduction in the number of chloroplasts in the cells of the yellow parts of the lamina. These parts are also thinner than the green parts on account of there being fewer cell layers (five cells as compared with seven) and smaller cells (27).

Geographical Distribution. The chlorotic disease has been recorded so far only from Worcestershire in England.

Control. The control methods for this disease are the same as for that caused by *Humulus Virus 1* (hop mosaic), but it must also be remembered that *Humulus Virus 3* is thought to be seed-transmitted.

HUMULUS VIRUS 4. Salmon and Ware

A new virus disease of the Fuggles hop is reported (30) from Sittingbourne, Kent. At present nothing is known of the virus, but the symptoms of the disease are described as follows. The bines of the affected hills were just past the bar-string, and their dead or dying tips were hanging away from the strings. One or two bines had succeeded in reaching the top wire. The lower leaves of the bine were down-curled, with very brittle petioles; the leaves of the laterals were mosaic-mottled. These symptoms are the same as those of mosaic (*Humulus Virus 1*) as it occurs in "Goldings" and the "Golding Varieties" of hops. A distinguishing feature is the late appearance of the disease in season and the resulting greater growth of the bine. It would appear, further, that this new virus cannot be the same as *Humulus Virus 1*, since it has been shown by numerous grafting experiments that the Fuggles hop can carry the former virus without showing symptoms of disease.

SANTALUM VIRUS 1. Coleman

Synonyms. *Sandal Spike Disease Virus* : *Sandal Spike Rosette Virus*, Rao and Iyengar, 1934.

The Virus and its Transmission. The problem of spike disease of sandal is one which has received a great deal of attention in India during the last decade, but it is still far from solution. There seems little doubt that the disease is due to a virus, since its infectiousness has been proved by grafting experiments. The virus is not transmissible by mechanical methods, by seed or by pollen.

In spite of a great deal of entomological study, the insect vector has not been identified, but some recent work (22) seems to suggest : (1) That the disease is actually insect-borne, (2) that the insect vector or vectors are nocturnal, (3) that the vector responsible may be one of the following : three types of Pentatomidae, two of Jassidae, and three of Fulgoridae.

Transmission of spike disease has been accomplished by various methods of grafting. Twig grafts which contain a large quantity of infective material always transmit the disease without exception whenever the grafts fuse with the stock. In all other methods of

inoculation, that is, budding, patch bark-grafts, ring bark-grafts, and leaf insertion, though organic connections are formed, the disease develops only in a certain percentage of cases, varying from 17 to 63 per cent, depending on the kind of graft used. This variation suggests that a minimum amount of the infective agent may be necessary to transmit the disease. There is also indication that some individual plants offer more resistance than others. The highest percentage of experimental transmission has been found among plants grafted in May and June, and the lowest in plants grafted in October. The virus is transmitted through the haustoria (5), but not as a rule by this method, in the case of young saplings 2 or 3 feet high, as their haustoria can attack only the small tender roots of other sandal, which in the case of spiked plants are all dead (40).

Diseases caused by *Santalum Virus 1*

Santalaceæ

Santalum album L. Sandal. *Spike disease.* The most striking feature of spike disease of sandal is undoubtedly the decrease in the size of the leaves, which is accompanied by a shortening of the internodes so that the leaves become very closely crowded together on the leaf-bearing branches. This does not appear all over the attacked tree at the same time, but shows itself on a few of the branches, the symptoms gradually spreading to the whole tree. At the same time the leaves display a tendency to stand out stiffly from the branch, thus giving rise to the somewhat spike-like appearance of the affected branches from which the disease derives its popular name. As the disease advances the fresh leaves produced become smaller and smaller. Thus, normal, fully developed leaves of sandal are about 3 inches long and 1 inch wide, whereas in the last stages of spike disease they have been found as small as $\frac{1}{3}$ inch long by $\frac{1}{16}$ inch wide. Another striking feature is the continuous growth in a tree or parts of a tree attacked by spike disease. There is none of the periodicity of growth usual in healthy trees, but fresh flushes of new leaves are produced throughout the year. This is accompanied by the formation of fresh branches, and as these leaves and branches are closely crowded together, a condition approximating to "witch's broom" is produced (see Fig. 81, A).

Another characteristic feature of the disease is the paler green colour of affected leaves; in later stages this becomes very marked, and in many cases the spike leaves take on a distinctly reddish

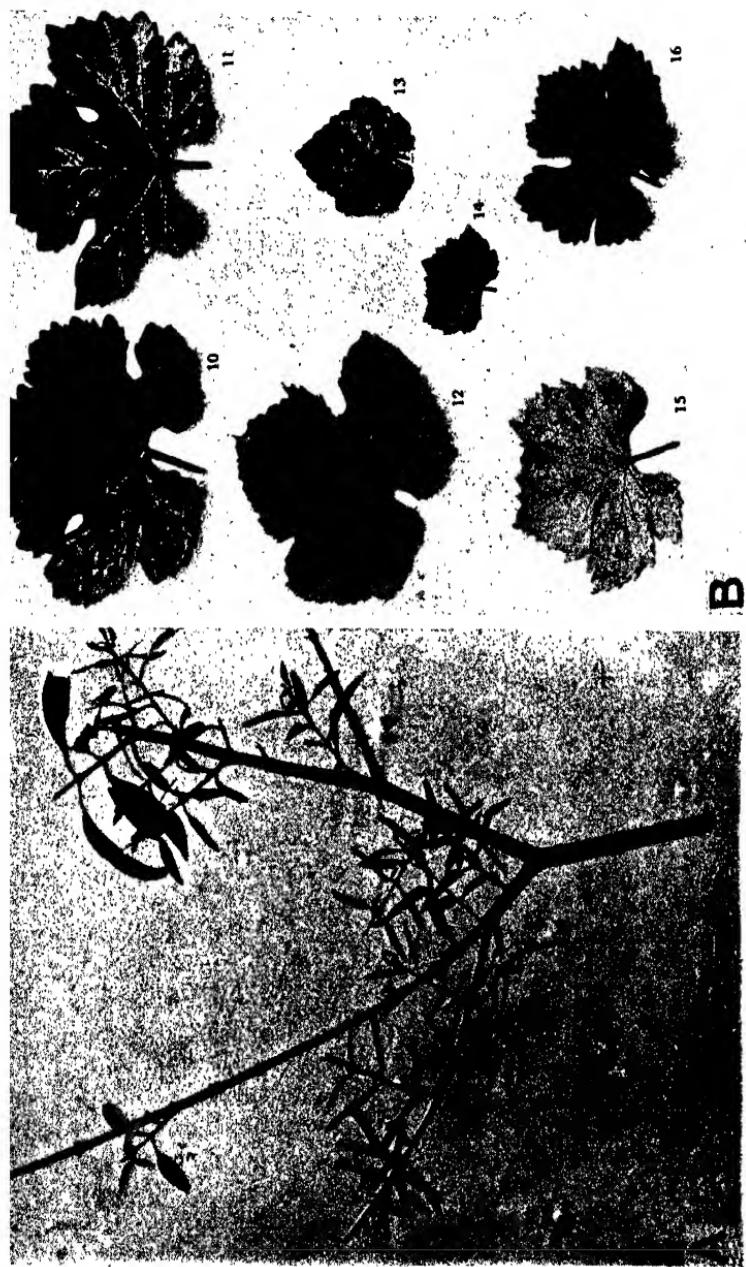


FIG. 31.
A. Sandal tree infected with *Santalum Virus 1* (sandal spike virus).
B. Vine leaves showing different types of mottling induced by *Vitis Virus 1* (vine mosaic virus).
(A, after Narasimhan; B, after Stranak.)

colour. Phyllody is another phenomenon connected with the disease. In the few cases where flowers have been observed on a spiked portion of a tree, this has always been in an early stage of the disease, and the flowers show almost invariably in place of the normal floral structures, a tuft of leaf-like bodies (*cf. Vaccinium Virus 1*, p. 206).

The roots of spiked trees are also affected, and in an advanced state of the disease the haustoria and the fine root ends are dead. As the sandal tree is a root parasite and probably obtains most, if not all, its nutriment from the soil solution indirectly through the roots of other trees by means of its haustoria, the death of these structures naturally involves the death of the sandal tree.

Histopathology. A characteristic symptom is the accumulation of starch in affected leaves and branches. Normal leaves show very little sign of starch deposition at any time, and in normal twigs there is only starch accumulation when the leaves are mature. In spiked leaves the starch is distributed throughout the parenchyma, and is especially pronounced in the sheaths of the fibrovascular bundles. The starch grains found in the leaves are minute, and are present, several together, in a chloroplast. In the twigs of spiked trees the starch occurs in large quantities as grains of considerable size in the pith, medullary rays, and wood and bast parenchyma.

Typical intracellular inclusions, or X-bodies, have been observed in sandal leaves affected with spike disease. They are generally round or oval and are often in close association with the nucleus. The inclusions have not been observed near the growing point and in the first pair of leaves, but can usually be found in the third pair. Measurements of the inclusion bodies along their greatest diameter show a variation ranging from 4.8 to 8.7μ (18).

Host Range. At present no other host plant for *Santalum Virus 1* is known, but certain plants in the sandal areas of India are commonly affected with an apparent virus disease with spike-like symptoms. Such plants are *Zizyphus ænolia*, *Dodonæa viscosa*, *Strachytarpheta indica* and *Vinca rosea*. Infected plants of these species have been grown in pot culture with healthy sandal, and although the haustoria of the latter fed on the roots of their hosts, no transmission was effected (89).

Geographical Distribution. The disease was first noticed in Coorg, India, in 1899, by McCarthy. In 1908 it was realised that spike was widely distributed in Mysore, Madras and North Coimbatore. The disease has travelled most rapidly towards

south and south-east India, though large tracts of country appear to have been passed over in this movement. Its progress towards the north has been very slow. In the sandal zone in southern India, while the disease has reached the southern limits, the northern half of the Mysore State and the adjacent sandal areas in Dharwar, Canara and north Coorg are still free of spike.

Control. Until the natural means of spread of *Santalum Virus 1* is discovered it is difficult to devise means of control. It has been observed that spike disease is much more prevalent in areas where undergrowth is abundant. Thus, the incidence of the disease is highest in scrub jungles and lowest in cultivated lands and private holdings. Among the types of undergrowth concerned, the presence of Lantana is suspected of being most favourable to the spread of infection, though thick undergrowth of other species in which Lantana is absent is also conducive to the spread of the disease. It is suspected that undergrowth favours the spread of the virus by harbouring the insect vector (39).

SANTALUM VIRUS 1A. Rao and Iyengar

Two types of sandal spike have been differentiated (41); the more common erect form of the disease just described has been designated "Rosette Spike," while the second is known as "Pendulous Spike." The latter is referred to here as caused by *Santalum Virus 1A*.

Disease caused by *Santalum Virus 1A*

In the pendulous type of sandal spike, the individual infected shoots show continuous apical growth and attain lengths of 1 to 3 feet, the leaves being confined to half to one-third the length of the growing tip. The growth in the length of the shoots being disproportionate to that in thickness, the infected twigs assume a conspicuous drooping habit. In the disease caused by the rosette or type virus, the spiked shoots are less than one-third the length of those in the pendulous type and grow stiff and erect. Dormant buds on spiked shoots of the pendulous type do not develop or grow at all, with the consequence that the excessive branching and the clusters of shoots characteristic of the rosette type of disease are absent.

The flowers are abnormal, with the buds considerably reduced in size and the pedicels elongated to three or four times the normal length. Anthers often show a reddish coloration and the pistil is

abnormally developed into a thick cylindrical body, with the top portion bent on itself in the bud. These flowers are sterile and do not form fruits. The root ends and haustoria do not die as in the disease caused by the type virus.

Santalum Virus 1A is transmissible by the same grafting methods as is the type virus, and similar intracellular inclusions are associated with both diseases.

SANTALUM VIRUS 2. Rao

Synonym. *Sandal Leaf-curl Mosaic Virus*, Rao, 1932.

The Virus and its Transmission. The virus is transmissible by ring bark-grafts, but not by mechanical sap-inoculation. The natural means of spread of the virus are not known.

Disease caused by *Santalum Virus 2*

Santalum album L. Sandal. *Leaf-curl mosaic*. In the field two stages of the disease are observed. In the first stage conspicuous mosaic spots develop between the veins of the leaves, which show slight rolling. Young leaves produced at the tips do not generally show mottling, but this appears as the leaves attain full size. In some of the pigmented types of sandal, a reddish-brown discolouration is seen at the edge of the leaves. The leaves retain their normal size, and the tree bears flowers and fruit as usual. The small branches and the leaves generally show a marked drooping habit. The trees appear to remain in this stage during one growing season.

In the second stage the new leaves show ruffling at the edges even when quite young, developing a wrinkled and mottled appearance as they grow older. Some of them become cup-shaped. Dwarfing of leaves and leaf-bearing twigs becomes conspicuous, and new leaves that develop become progressively smaller in size, and finally get curled down the mid-rib. The lower leaves in the shoots generally curve inwards, while those at the tip bend outwards. The colour of the leaves is green to pale green, turning greenish-yellow towards maturity. The reduction in leaf blade, unlike that in spike disease, is generally greater in length than breadth, and sometimes more or less uniform both ways. The length of the internodes and petioles, however, is not appreciably reduced, though there is a considerable reduction in thickness. The leaves lose their flexibility, become thickened and brittle and fall off prematurely.

The disease has been recorded from Bangalore City and Mysore City in India (38).

VITIS VIRUS 1. Stranak

Synonyms. *Vine Mosaic Virus*: possibly the same as *Roncet*, *Court-noué*, *Rougeau*, *Reisigkrankheit*, etc.

The Virus and its Transmission. Later investigation may show that more than one virus is concerned in the production of vine mosaic, but for the present the evidence is not sufficient to justify such a conclusion.

According to Stranak (36) the virus is easily transmissible by sap-inoculation methods and is also carried by the pruning knife. He states that the insect vector is the scale insect *Lecanium corni* and some undetermined species of aphides. There is no information on the properties of the virus.

Disease caused by Vitis Virus 1

Ampelidaceæ

Vitis vinifera L. The Vine. The mosaic mottling induced by the virus in the vine is of a very varied character. In one form the veins are either partly or completely picked out in yellow on the dark green background of the leaf. The mottling may consist of very numerous small yellow dots on the leaf, or, alternatively, of large pale areas interspersed with bright green patches. In some cases the leaves may be entirely yellow or almost white without mottling. Certain varieties show a severe deformation of the leaves which are crimped between the main veins. Many of the affected stocks show very poor growth (see Fig. 31, B).

There is some evidence of the existence of symptomless virus carriers in certain varieties of vines, White Riesling for example.

Histopathology. In plants severely affected the cordons first described by Petri (21) for the disease known as "roncet" can easily be distinguished. These are special intracellular formations of a nature similar to the cell walls which form solid cordons traversing the cells in a given direction. These bodies develop first in the cambium and then in the wood and cortex.

The chloroplasts are attacked in a characteristic manner, at first in the palisade tissue. In cases of severe infection the chlorophyll granules are disintegrated and the products of their degeneration fill up the conducting elements. Stranak states that numerous small granules can be detected in the disintegrating

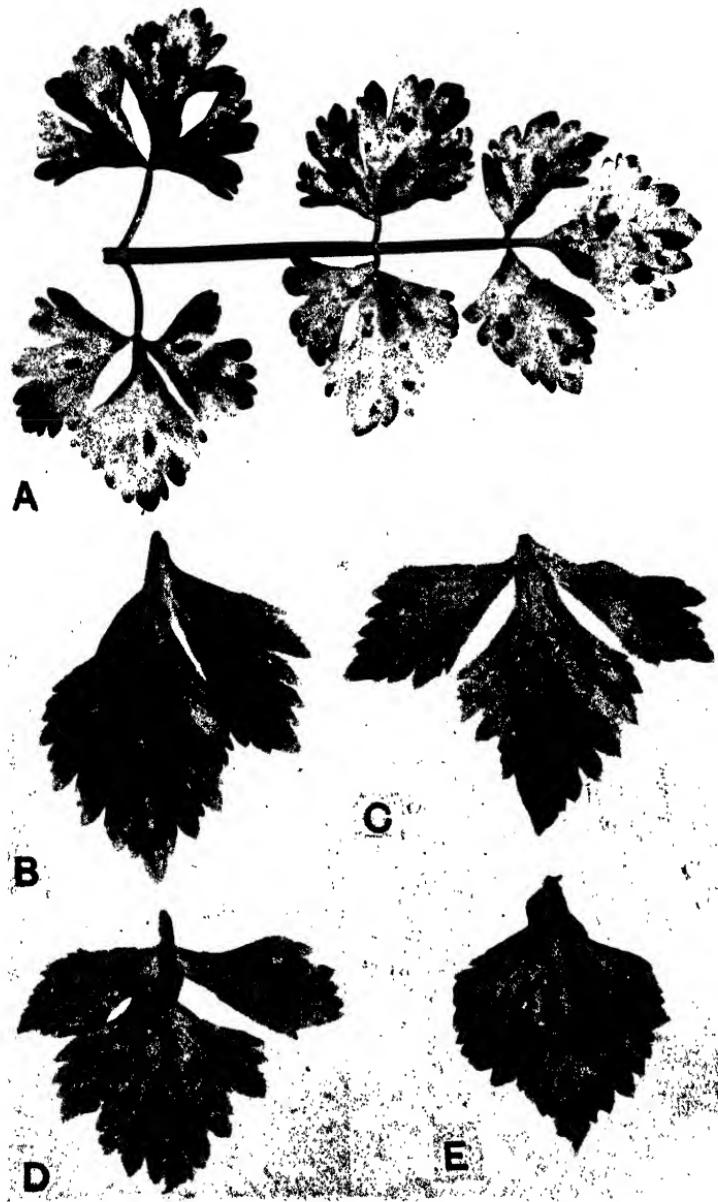


FIG. 32.

A. Celery leaf infected with *Apium Virus 2* (celery calico virus).
B-E. Leaves of celery plant infected with *Apium Virus 1* (western celery mosaic virus), showing varying types of mottling.
(After Severin and Freitag.)

chloroplasts, in the cytoplasm, and even in the nuclei of affected plants. These granules vary in size from 1 to 3μ .

Geographical Distribution. The mosaic disease of the vine is probably almost world-wide in its distribution ; it seems to occur wherever grapes are grown. It has been recorded from France, Italy, Bulgaria and Czechoslovakia.

Control. No very efficient methods of control for vine mosaic have yet been discovered and only general recommendation can be made. All obviously and severely affected vines, together with their roots, should be removed and destroyed. A good insecticide for the destruction of possible insect vectors should be used, and grafting knives should be sterilised each time before being used on a fresh vine.

APIUM VIRUS 1. Severin and Freitag

Synonym. *Western Celery-mosaic Virus*, Severin and Freitag, 1935.

The Virus

The following information on the properties of this virus was kindly supplied by Dr. Severin.

Resistance to Alcohol. In a suspension the virus in the supernatant fluid withstands treatment with 30 per cent alcohol for one hour, while the precipitate withstands 40 per cent for a similar period.

Thermal Death-point. The thermal inactivation point is 55°C . in ten-minute exposures.

Dilution End-point. The tolerance to dilution of the virus is 1 : 4,000.

Resistance to Ageing. Inactivation of the virus occurs after expressed sap of diseased celery plants is exposed to the air at room temperatures for seven days. If the sap is kept at -18°C . infectivity is not lost after six months.

Filterability. The virus passes all grades of Chamberland filters.

Transmission. The virus is sap-inoculable, but the insect vector does not appear to be known.

Disease caused by Apium Virus 1

Umbelliferæ

Apium graveolens L. Celery. *Western Celery Mosaic*. *Apium Virus 1* causes a bold mosaic mottling in the leaves of affected

celery. The leaflets show cleared veinlets or a pronounced mottling (see Fig. 32, B-E).

Geographical Distribution. This virus has only been recorded from California (33).

APIUM VIRUS 2. Severin and Freitag

Synonym. *Celery Calico Virus*, Severin and Freitag, 1935.

The Virus and its Transmission. There is no information available at the moment on the properties of this virus, but the insect vector appears to be *Aphis gossypii* (see p. 515).

Disease caused by Apium Virus 2

Umbelliferae

Apium graveolens L. Celery. *Calico disease*. Affected celery plants exhibit a striking chlorosis in which the leaves show green islands of tissue in the lemon-yellow areas (see Fig. 32, A).

Geographical Distribution. *Apium Virus 2* has only been recorded from California (33).

VACCINIUM VIRUS 1. Dobroscsky

Synonyms. *Cranberry False-blossom Virus*; *Wisconsin False-blossom Virus*.

The Virus and its Transmission. The virus is not sap-inoculable, and it has not been transmitted by grafting owing to the difficulty of achieving organic union in the attempted grafts. There is no information on the physical properties of the virus. The insect vector is *Euscelis striatulus* Fallen, the blunt-nosed leathopper (see p. 477).

Disease caused by Vaccinium Virus 1

Ericaceae

Vaccinium macrocarpon Ait. Cranberry. *False-blossom*. The disease caused by the virus in cranberry is most readily recognised when the plant is in bloom. The name "false-blossom" is particularly appropriate to the disease, as the flowers show the symptoms most clearly and are usually rendered sterile. The flower, as a whole, assumes an upright position instead of the normal curve of the pedicel. The calyx lobes of diseased flowers become enlarged, the petals are short and streaked with red and green, and the stamens and pistils are more or less abnormal. When the disease is severe the entire flower may be replaced by successive whorls of leaves or by a short branch. The leaves also



FIG. 38. *Vaccinium Virus 1* (cranberry false-blossom virus).

- A. Cranberry false-blossom disease ; note witch's broom symptoms.
- B. Fruiting upright from a three-year-old cranberry plant with two false-blossom buds ; note the leaf growing from the centre of the upper bud.
(After Dobroscky.)

show symptoms of the disease. Axillary buds which are usually latent produce numerous negatively geotropic branches with many crowded leaves which are closely appressed to the stem. The resulting structure is referred to as a "witch's broom." In autumn the leaves take on a reddish hue, which enables a diseased area to be located at some distance. The cranberry sets its fruit buds for the succeeding year in autumn. In a diseased plant these terminal fruit buds are enlarged and in an advanced stage of development. As a result, the diseased buds are frequently killed during spring frosts. One of the most striking symptoms of the disease results from the production of shoots by axillary buds which in a healthy plant would remain dormant (see Fig. 33).

Under certain conditions diseased plants mature their fruits, but such berries are usually small, misshapen, and held in an erect position. Sections of these berries generally show only a few seeds, most of the ovules remaining undeveloped. Such seeds as are produced are generally abnormal; some are not true seeds at all, since they apparently contain no embryo or endosperm.

The symptoms of the disease on seedlings are more difficult to recognise, but as a rule the numerous negatively geotropic shoots with small closely appressed leaves are sufficient for a correct diagnosis to be made (7).

Host Range. Only one other species of *Vaccinium*, *V. oxycoccus*, the small European cranberry, appears to be susceptible to infection with the virus.

Geographical Distribution. The virus is thought to have been noticed for the first time in Wisconsin, U.S.A., in 1885, and was first identified in Massachusetts in 1914. At present it occurs wherever cranberries are grown commercially in the United States of America east of the Mississippi River. It has recently been found in Canada. The virus has not been recorded from any other part of the world.

Control. Since the leafhopper *E. striatulus* seems to be the only agent for the spread of false-blossom, the disease will be controlled by the destruction of this insect. One of the chief methods of insect control on wet bogs, that is, bogs which have a reservoir, has been to flood the bogs for a long or short period as the case may demand. For the control of certain insects the bog is submerged for twenty-four hours, after which the water is drawn off. To ensure the destruction of all the insects the trash and bordering plants are burned with kerosene oil. When a bog is badly infested, the grower holds the winter water flow on the bogs

until July, thereby killing all insects at the cost of that season's crop.

Some success has been achieved by means of spraying, and a mixture of pyrethrum and soap has proved to be the most effective insecticide.

If a bog is badly diseased, the only effective measure of control is to burn the old vines and replant. The fact that some varieties of cranberry are more resistant than others to *Vaccinium Virus 1* points the way for future progress. Fracker (9) has shown that the McFarlin variety is the most resistant to the disease.

DAHLIA VIRUS 1. Brandenburg

Synonyms. *Dahlia Mosaic and Yellows Virus*, Howe, 1922; *Dahlia Stunt or Dwarf Virus*, Howe, 1923; *Dahlia Runting Virus*, Connors, 1926; *Dahlia Leaf-curl and Rosette Virus*, Martin, 1929.

The Virus and its Transmission. The virus is not sap-inoculable, and in consequence there is no information on its physical and other properties. It is transmissible by grafting, but is not carried in the seed. The insect vector is the aphid *Myzus persicae* Sulz. (see p. 538).

Differential Hosts

The American variety of dahlia, Robert Scott, develops areas of necrosis on the mid-veins when infected with this mosaic virus. These areas commonly appear about midway in the length of individual pinnae and bring about a sharp downward turning of the pinna at that point. This vein necrosis, together with the characteristic dwarfing that accompanies it, is a valuable diagnostic character. The fact that this virus is not transmissible to Solanaceous plants differentiates it from *Lycopersicum Virus 3*, which also produces mosaic symptoms on the dahlia.

Diseases caused by Dahlia Virus 1

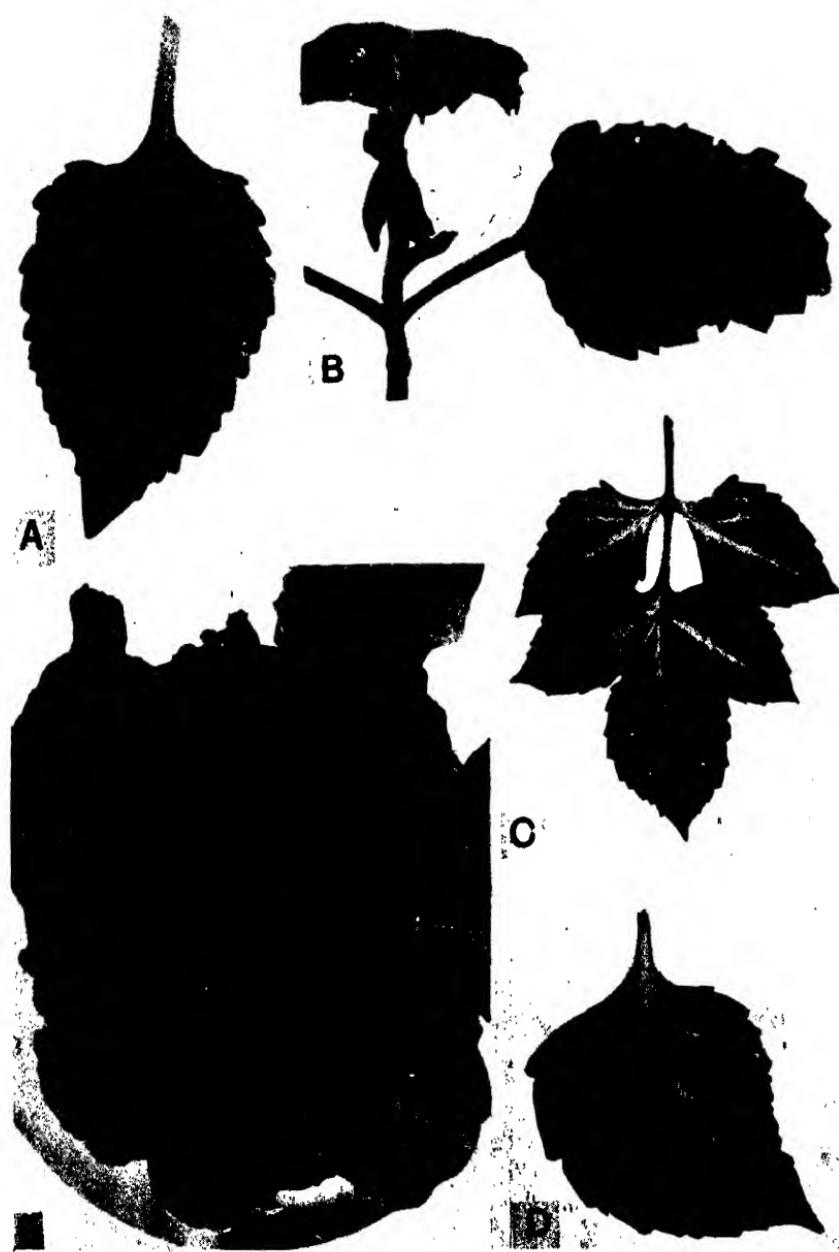
Compositæ

Dahlia variabilis Desf. Dahlia. *Dahlia mosaic*. The most characteristic symptom of dahlia mosaic is "vein-banding." The

FIG. 34. A-D. *Dahlia Virus 1* (dahlia mosaic).

- A. Dahlia, var. White Empress, natural infection.
- B. Dahlia, unknown variety, natural infection.
- C. Dahlia, var. Jersey's Beauty, natural infection.
- D. Dahlia, var. Grace Ricards, natural infection.
- E. Lettuce plant, showing symptoms caused by *Lactuca Virus 1* (lettuce mosaic virus).

(A, C and D, after Brierley.)



normal green colour develops irregularly in the mosaic leaf, bands adjacent to the mid-rib or branch veins remaining yellowish-green or pale green when the remainder of the leaf has reached normal colour. The width and form of this banding vary in different varieties and to a less extent in individual plants of a given variety. The typical colouring of the bands is uniform within a variety, but varies among different sorts from a green only slightly paler than normal to a bright yellow. As the affected leaf grows older the discoloured areas tend to approach the normal green of the remainder of the leaf and the colour pattern may become masked. Vein-banding is the most reliable diagnostic symptom of dahlia mosaic and it appears in varieties which show no distinctive distortion, necrosis or dwarfing (see Fig. 34).

In certain American varieties, such as Snowdrift, leaves of mosaic plants may become generally yellowed, the margins roll upward on the mid-ribs, and the lateral pinnæ are more or less twisted. Vein-banding may be poorly expressed in this variety. Some varieties show marked distortion rugosity and blistering of the leaves (see Fig. 34, B). Masking of the chlorotic symptoms during growth is a frequent phenomenon.

Shortening of the internodes is prominent in mosaic plants of all those dahlia varieties which are intolerant of the virus. Accompanying this shortening of the main stem is a tendency to force the lateral shoots, which are in turn shortened, producing the familiar short, bushy, "crippled" habit designated as "stunt." The flower stems are also short in such varieties, so that it is often impossible to cut a flower with suitable stem length.

The roots of intolerant varieties may also tend to be shorter than normal, but this character is not regarded as of much diagnostic value.

The flowers of affected plants are usually normal and show no change or "breaking" in the colour (4).

Histopathology. Intracellular inclusions, or X-bodies, have been described by Goldstein (11) in the tissues of mosaic-infected dahlias. The bodies may be rounded or oval, or extremely elongated. The elongated bodies usually occur in elongated cells, although similar cells may contain minute or medium-sized rounded or oval bodies. Small cells usually contain small inclusions. Often the X-bodies lie in close proximity to the nucleus of the cell, pressed up against it, or partially surrounding it.

Differences in Varietal Reactions to Dahlia Virus 1. The symptom picture due to this virus varies considerably according

to the variety of dahlia affected. Brierley (4) provisionally classifies these different diseases into five types, as observed on a selection of American dahlias : (1) *Mild Mosaic*, affected plants of Mrs. I. de Ver Warner, Jersey's Beauty, Faith Slocombe, and Le Toreador show vein-banding, but no evident dwarfing or distortion ; (2) *Rugose Mosaic*, diseased plants of Calizona and other varieties show vein-banding together with rugose or blistered leaves ; (3) *Rugose Rosette*, affected plants of Casper G. Ware, Mrs. M. W. Wilson, Doris Wilmore and others combine the symptoms of types (1) and (2) with marked rosetting ; (4) *Yellow-top Rosette*, certain plants of Snowdrift, White Empress and a few other varieties show yellowing of the upper leaves and marked dwarfing without evident mottling ; (5) *Dwarf*, affected plants of Robert Scott show very severe dwarfing without mottling. Of these types of symptoms, type (1) may be described as tolerant, type (2) as semi-tolerant, and types (3), (4) and (5) as intolerant.

Host Range. No plant outside the genus *Dahlia* has been experimentally infected with *Dahlia Virus 1*, but all members of this genus seem to be susceptible, including *D. maxonii* Safford and *D. imperialis* Roezl.

Geographical Distribution. *Dahlia Virus 1* seems to be widely distributed ; it has been recorded from most of the States of the U.S.A. It is present in Holland, in Germany and in England. The writer has frequently observed the disease in commercial stocks of dahlias in England where the virus seems to be on the increase.

Control. The soundest method of control of dahlia mosaic consists in growing and multiplying selected healthy stocks at a reasonable distance from other, possibly infected, dahlias. Roguing infected plants from stocks which are largely healthy is a useful measure when practicable, but there is the difficulty of recognising the symptoms, especially in some of the tolerant varieties which may approximate to the "carrying" condition and yet be dangerous sources of infection.

Control of the insect vector by greenhouse fumigation during the period when cuttings or seedlings are grown under glass is both feasible and important.

The use of tolerant varieties only as a way out of the difficulty is not recommended, since it restricts the choice of varieties and because even these are themselves adversely affected as compared with healthy plants (4).

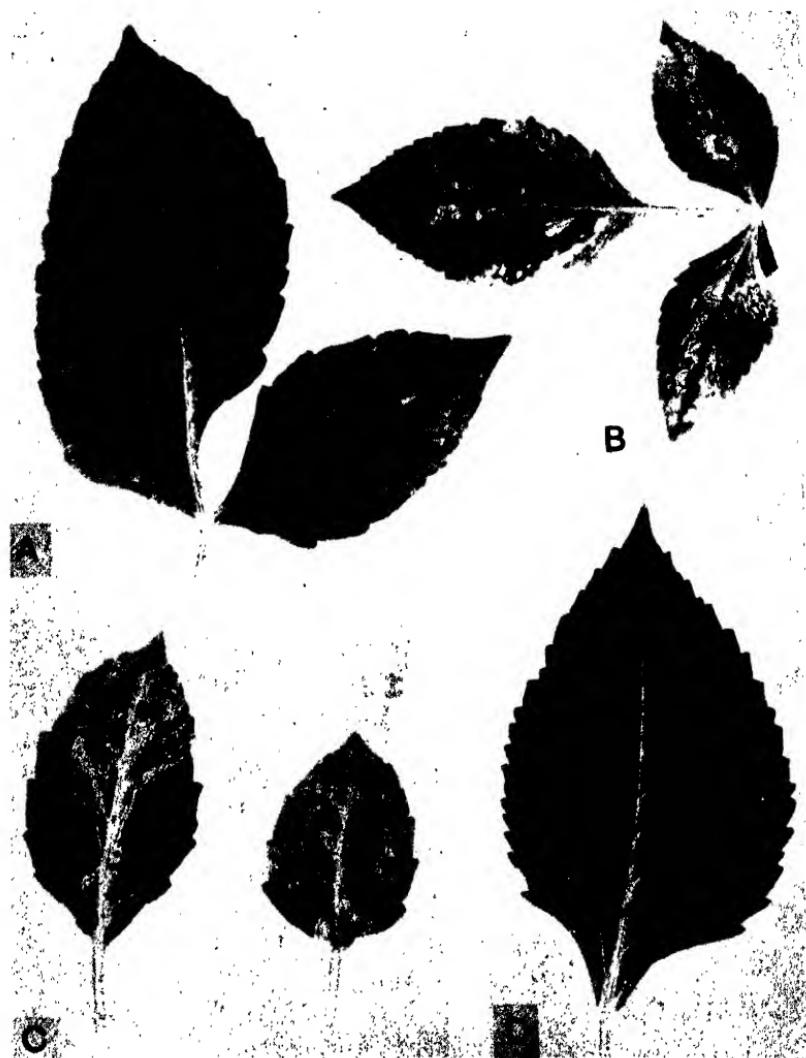


FIG. 35.

- A. Dahlia, var. Catherine Wilcox, affected with *Dahlia Virus 2A* (yellow ringspot virus).
- B. Dahlia, var. Long Island, naturally infected with *Dahlia Virus 2A*.
- C. Dahlia, var. Chautauqua Salute, naturally infected with *Dahlia Virus 2* (dahlia ringspot virus).
- D. Dahlia, var. Calvin Coolidge, Jr., naturally infected with *Dahlia Virus 3* (oakleaf virus).

(After Brierley.)

DAHLIA VIRUS 2. Brierley

Synonym. *Dahlia Ringspot Virus*, Brierley, 1933.

The Virus and its Transmission. It is assumed that this virus which causes a ringspot disease in dahlia is a different virus from *Lycopersicum Virus 3* (tomato spotted wilt virus) which also causes a ringspot disease in this plant (see p. 301). *Dahlia Virus 2* is not apparently transmissible by mechanical methods, and it is not carried by the aphid, *Myzus persicae*; the insect vector is not known.

Disease caused by Dahlia Virus 2

Compositæ

Dahlia variabilis Desf. Dahlia. *Dahlia ringspot* (see Fig. 35, C). The symptoms appear as irregular concentric rings or irregular zigzag markings, intricate hieroglyphic patterns, and green islands. The width and number of zones expressed vary within a given variety. The colour of the chlorotic areas varies from pale green to yellowish-green in different varieties, but is relatively uniform within a variety. In field plants ringspot symptoms may first be expressed as a chlorotic pattern which later becomes necrotic. In some varieties only chlorotic symptoms have been observed. Stunting or distortion is not characteristic of this disease.

Geographical Distribution. This disease was first observed by Connors, in 1929, in New Jersey, U.S.A., and it has been recorded in New York and Connecticut. There is no information on this disease in Europe.

DAHLIA VIRUS 2A. Brierley

Synonyms. *Dahlia Yellow Ringspot Virus*, Brierley, 1933; *Dahlia Virus 2B*, J. Johnson's classification.

The Virus and its Transmission. Little is known of this virus, and it is dealt with here as a strain of *Dahlia Virus 2*. It does not appear to be sap-inoculable.

Disease caused by Dahlia Virus 2A

Compositæ

Dahlia variabilis Desf. Dahlia. *Dahlia yellow ringspot* (see Fig. 35, A and B). The symptoms in the variety Long Island are bright yellow concentric rings and zigzag patterns. In other dahlia varieties, such as Catherine Wilcox or Robert Scott, the symptoms are less marked and consist of yellow rings or segments

of rings mostly near the tips of the leaflets. The disease can be differentiated from that caused by the type ringspot virus by the prominent yellow patterns as contrasted with greenish and yellowish-green patterns (4).

Geographical Distribution. The virus has only been recorded from Utah, U.S.A.

DAHLIA VIRUS 3. Brierley

Synonym. *Dahlia Oakleaf Virus*, Brierley, 1933.

The Virus and its Transmission. Very little is known of the "oakleaf" disease and the virus causing it. The latter is included here as a separate entity on the evidence of symptoms only (4), and much more work is needed before it can be definitely classified as a new virus.

Disease caused by Dahlia Virus 3

Compositæ

Dahlia variabilis Desf. Dahlia. *Oakleaf disease* (see Fig. 35, D). The symptoms of which the name "oakleaf" is descriptive are often well marked in the American variety of dahlia Calvin Coolidge, Jr. The chief symptom is the development of a pale chlorotic line across the middle of the leaf, somewhat suggestive of the outline of an oakleaf. The disease is not known to involve dwarfing, distortion or necrosis. The appearance of rather poorly defined large chlorotic rings seems also to be associated with the disease. Oakleaf has only been recorded from the United States of America.

CALLISTEPHUS VIRUS 1. Kunkel

Synonyms. *Aster Yellows Virus*; *New York Aster Yellows Virus*; *Lettuce White-heart Virus*; *Lettuce "Rabbit-ear" Virus*; *Lettuce*; *Rio Grande Disease Virus*.

The Virus

Since this virus is not sap-inoculable there is at present no information on its physical and other properties.

Transmission. The virus can only be transmitted by grafting and budding and by the agency of one or two species of leafhoppers (Jassidae). The most efficient insect vector is *Cicadula divisa* Uhl. (= *sexnotata* Fall.) (see p. 472), but the following two species also transmit a Californian strain of the virus, *Thamnotettix montanus* Van D., the mountain leafhopper, and *T. geminatus* Van D.

The relationship between this virus and the insect *C. divisa* is of great interest. Kunkel (14) has shown that there is a delay of ten days in the development of infective power within the insect. Since this period is usually longer than the nymphal life of the vector, it follows that the nymphs are unable to transmit the virus, although they are able to imbibe it from the diseased plant. Adults, however, arising from viruliferous nymphs, can transmit the virus to a healthy susceptible plant without further access to a source of infection. If, however, the development of viruliferous nymphs be retarded by exposure to low temperatures beyond the "incubation period" of the virus, then the nymphs can transmit the disease. This phenomenon affords an interesting contrast to the transmission of *Lycopersicum Virus 3* (tomato spotted wilt virus) by thrips. In the latter case the adult insect is apparently unable to imbibe the virus and it is necessary for the virus to be taken up by the larval thrips before it can be transmitted by the adult.

The other two leafhoppers, *Thamnotettix montanus* and *T. geminatus*, are vectors of a Californian strain of this virus (*Callistephus Virus 1 A*), often known as "celery yellows." In experimental infections, *T. montanus* transmitted the virus to 2·9 per cent of the asters and 26·1 per cent of the celery plants. *T. geminatus* failed to transmit the virus to or from asters, but infected 18·7 per cent of the experimental celery plants (32).

Differential Host

Callistephus chinensis Nees. China Aster. The disease produced in the China aster by this virus is more fully dealt with on p. 217, in the description of the diseases produced in plants belonging to the Compositæ, but some of the more typical symptoms are mentioned here. The first symptom is a clearing of the veins of the young leaves, and this is followed by a general chlorosis, not a mottling, of the new leaves. Petals which normally contain no chlorophyll frequently become quite green when diseased. Another striking symptom results from the abnormal production of secondary shoots. Instead of lateral buds remaining dormant, they produce long, thin, chlorotic branches. The main symptoms then are diffuse and well-marked chlorosis, clearing of veins, occasional one-sided or sectorial infection, upright habit of growth, malformation and increased growth in certain organs, but dwarfing of other organs, dwarfing of the plant as a whole, and the abnormal production of secondary shoots (15).

Diseases caused by Callistephus Virus 1

Ranunculaceæ

Adonis aestivalis L. Affected plants are dwarfed and chlorotic and produce many secondary shoots after having the disease for a considerable period of time. The leaves of infected plants are smaller and shorter than those of healthy plants.

Papaveraceæ

Papaver nudicaule L. Diseased poppies are severely stunted and show the chlorosis which is so typical of infection with this virus.

Cruciferæ

Cheiranthus Allionii Hort. Affected plants are chlorotic and abnormally branched. The leaves are longer and narrower than the leaves of healthy plants.

Malcolmia maritima R. Br. Virginia stock. Diseased plants show stunting, but very little chlorosis. They produce many upright-growing secondary shoots. They bear virescent flowers from the pistils of which long stems arise, these in turn bear secondary virescent flowers.

Radicula sylvestris (L.) Druce. Affected plants are dwarfed and produce many slightly chlorotic secondary shoots.

Polygonaceæ

Fagopyrum esculentum Gaertn. Buckwheat. Symptoms of infection on buckwheat are slight chlorosis and dwarfing, with indefinite proliferation of the flower buds and abundant production of small greenish flowers on long, rather erect pedicels. Numerous flowers are produced in the leaf axils.

Chenopodiaceæ

Spinacia oleracea L. Spinach. The first sign of infection is a clearing of the veins of the younger leaves. Many upright secondary shoots are sometimes produced and the plants are dwarfed and slightly chlorotic.

Amaranthus caudatus L. and *A. auroro*. Stunting and chlorosis, with the production of numerous short, secondary shoots, are characteristic of infection with *Callistephus Virus 1*. The leaves show clearing of the veins.

The virus causes the leaves of *A. auroro*, which are normally red, to become yellowish-grey.

Umbelliferæ

Daucus carota, var. *sativa*. Carrot. Carrot plants naturally infected with *Callistephus Virus 1* show a marked yellowing of the younger central leaves, while the outer older leaves are usually reddish or purple. The discolouration of the older leaves may, however, fail to develop under glasshouse conditions. The younger central leaves are dwarfed and the petioles are sometimes twisted; occasionally a dense growth of adventitious, chlorotic shoots develops at the centre of the crown. The leaflets on the shortened petioles are sometimes reduced to short filaments, which often become dry. Carrot plants experimentally infected with *Callistephus Virus 1* sometimes develop a short central seed stalk or several seed stalks, and a constriction may occur below the crown of the carrot. Plants in an advanced stage of the disease show numerous bunched rootlets arising from elevations on the carrot root.

Pastinaca sativa L. Parsnip. Affected plants of the cultivated parsnip are dwarfed and chlorotic, and the innermost leaves have twisted petioles. There are many secondary shoots and the leaves are smaller than normal.

Petroselinum hortense, var. *radicosum*. Hamburg or turnip-rooted parsley. A dense growth of chlorotic leaves arises at the centre of the crown. These leaves are dwarfed with upright petioles which are frequently twisted. The incubation period of the disease varies from 36 to 106 days, with an average of 65·2 days.

Compositæ

Callistephus chinensis Nees. China aster. *Aster yellows* (see Fig. 36). Kunkel (14) describes the yellows disease of aster as follows: aster leaves affected with yellows never show mottling. The disease is therefore easy to distinguish from chloroses of the mosaic type. In advanced stages the virus is always systemic in the above-ground portions of the plant. The first symptom to be observed on a young plant is a slight yellowing or clearing of the veins in the whole or part of a single young leaf. After a plant has been diseased for some time the new leaves are chlorotic throughout. One-half or more, or a sector amounting to less than one-half of a plant, may be chlorotic for some time before the remaining portion is affected. This shows that rapid spread round the stem of the plant does not take place in many cases. When the attack is severe the young leaves are almost white. Such leaves may become more or less green as they grow old. The disease causes

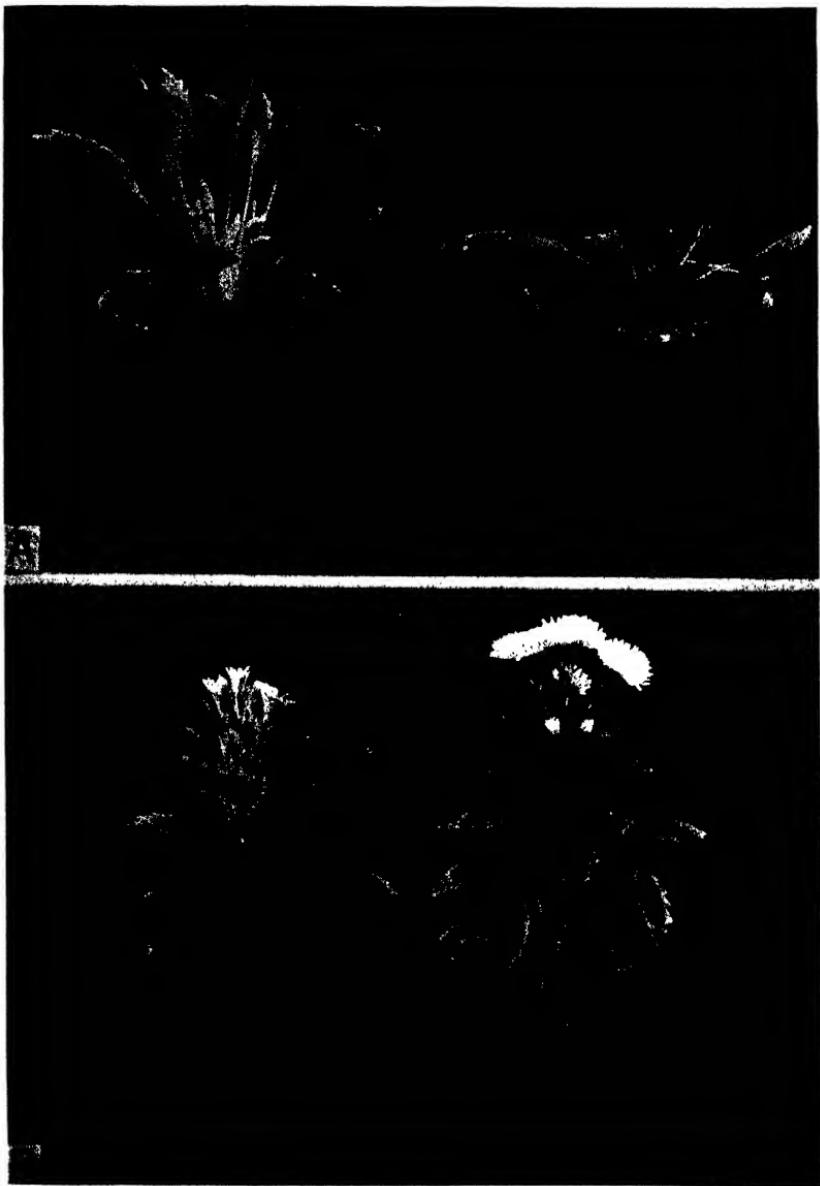


FIG. 36. *Callistephus Virus 1* (aster yellows virus).

A. (Left) Young aster plant with yellows. (Right) Healthy plant of same age, var. Semple's Shell Pink.
B. (Left) Aster plant with yellows. (Right) Healthy plant, var. Semple's Shell Pink.

(After Kunkel.)

chlorosis in all green portions of the plant. Strangely enough, however, petals which normally contain no chlorophyll become quite green when diseased. While the virus depresses chlorophyll-production in portions of the plant that are normally green, it causes the production of some green coloured substance in floral parts where chlorophyll is not normally present.

In different plants and under different conditions the disease causes widely different degrees of fading or yellowing of leaves, varying from slight to extreme chlorosis. Some aster plants show much less chlorosis and are much less stunted than others.

One of the most striking symptoms of the disease results from the production of large numbers of secondary shoots. Such shoots frequently arise in the axils of leaves that show a normal green colour and were mature before the plant became infected. They are always thin and have the appearance of etiolated branches. The degree of stunting varies with the age of the plant at the time it becomes infected and with the size of the sector infected. The effect of the disease on the main stem is greatly to shorten the internodes. It usually has the opposite effect on the secondary shoots. Diseased plants and seeds are often much larger than healthy ones, but cases also occur in which they are dwarfed. The flower heads are always more or less dwarfed. Individual flowers in some cases develop into vegetative branches which may or may not bear small flower heads. Trichomes on diseased flowers frequently develop into leaf-like structures. The root systems of diseased plants appear normal, but are smaller than those of healthy plants.

Another interesting result of the yellows disease is the change which it brings about in the response of the plant to gravity. Instead of diseased leaves lying flat and making a broad angle with the vertical, they stand upright.

There are also certain morphological changes. Diseased leaves are frequently somewhat deformed. Their petioles are longer than those of healthy leaves of the same age. The leaf blade, on the other hand, is narrower, and, on the whole, smaller than the normal blade. Diseased leaves may have deep clefts and notched margins, but are seldom severely deformed. Necrosis is a symptom of advanced stages of the disease. It is most severe in the stem tissue, a short distance below the apical bud of the main stem or of a branch. It causes the collapse of certain tissues, but does not kill the plant.

Zinnia elegans Jacq. The zinnia appears to be resistant to

infection with *Callistephus Virus 1*, and this is important in view of the fact that zinnia is susceptible to infection with the Californian strain of this virus (*Callistephus Virus 1A*).

Zinnia multiflora L. Unlike the foregoing species, this plant is susceptible to infection. Affected plants are chlorotic with numerous upright secondary shoots.

Cineraria hybrida Hort. Diseased plants are chlorotic and dwarfed. They produce many short upright-growing secondary shoots and leaves with long petioles. Clearing of the veins is an early symptom.

Chrysanthemum maximum Ramond. Shasta daisy. Diseased plants show a general chlorosis and upright habit of growth. Clearing of veins appears in some leaves. Most specimens are only partly diseased.

Gaillardia aristata Pursh. Affected plants are badly dwarfed and chlorotic, with many side shoots bearing small upright leaves.

Calendula officinalis L. The flowers of this species are affected in much the same way as are those of the aster, and the usual dwarfing and chlorosis are also present.

Tagetes erecta L. African marigold. Plants are dwarfed and chlorotic and secondary shoots develop earlier than in normal plants, but are not more numerous. Plants never become bushy and the habit of growth is not noticeably changed. Infection at an early stage of growth prevents the formation of blossoms.

Lactuca sativa L. Lettuce. Affected plants of lettuce are dwarfed and chlorotic. They fail to make heads, but produce many upright secondary shoots. The margins of diseased leaves often show brown coloured pustules which are old latex clots. Flowering side branches are greatly shortened. Diseased plants of Romaine lettuce are chlorotic and fail to set heads.

Taraxacum officinale Weber. Dandelion. Chlorosis and the production of numerous secondary shoots are characteristic of infection in the dandelion. The leaves are reduced in width, show clearing of the veins and a more upright habit of growth than is usual with normal plants. One of the most striking symptoms on the dandelion is the bronzing or reddening of the leaves. The flower heads are affected much as they are in the aster.

Solanaceæ

Solanum tuberosum. Potato. The potato plant appears to be immune from infection with *Callistephus Virus 1*, and this is another point of difference between the type virus and the

Californian strain (*Callistephus Virus 1A*), which does infect potato.

Lycopersicum esculentum L. Tomato. The abnormal production of secondary shoots is one of the most common symptoms of infection with *Callistephus Virus 1*, and it stimulates growth in buds which would remain dormant in healthy plants. In the tomato the virus not only causes this production of abnormal secondary shoots, but it stimulates the development of leafy buds and shoots in the axils of leaflets as well. The plants show, in addition, a certain amount of chlorosis and a bushy type of growth. They are considerably dwarfed. It appears that the virus is not easily transmitted to the tomato by the insect vector (*Cicadula divisa*).

Petunia hybrida Vilm. Affected plants are chlorotic and badly dwarfed; they bear malformed virescent flowers. There is much branching and rosetting of secondary shoots.

Browallia demissa L. In diseased plants of this species the chlorosis is only slight; dwarfing, however, is very pronounced.

Scrophulariaceæ

Mimulus luteus L. Monkey flower. Plants are somewhat dwarfed and slightly chlorotic. The stems are not more than half as thick as those of healthy plants. Many slender secondary shoots are produced. Large numbers of aerial roots grow from the nodes.

Calceolaria spp. Diseased plants are chlorotic and much dwarfed. Secondary shoots remain small and leaves are much reduced in size.

Veronica peregrina L. Symptoms consist of chlorosis and the production of many secondary shoots.

Gesneriaceæ

Gloxinia spp. Chlorosis is slight and many secondary shoots are produced. Diseased plants do not produce flowers.

CALLISTEPHUS VIRUS 1A. Severin

Synonyms. *Celery Yellows Virus*, Severin; *Californian Aster Yellows Virus*, Severin.

The Virus and its Transmission. The distinction between this strain and the type virus seems to be largely a question of differences in the host range. There seem also to be two additional

insect vectors besides *Cicadula divisa* (*sexnotata* Fall.). These are *Thamnotettix montanus* Van D. and *T. geminatus* Van D. (32).

Diseases caused by Callistephus Virus 1A

The following host plants of this strain are apparently resistant to infection with the type virus.

Umbelliferæ

Apium graveolens. Celery. *Celery yellows*. The first symptom of the disease to appear in celery grown in the glasshouse is the development of a vertical or upright position of the petioles, which are somewhat longer than those of healthy leaves of the same age. The petioles of the innermost or youngest leaves are shortened and chlorotic, and begin to twist and intertwine. There may even be a complete circular twist of the petioles. A general yellowing of the plant then develops with a premature blanching of the outer leaves. The vertical or upright petioles gradually assume a flat position. The petioles are brittle, break easily, and often crack. In the later stages of the disease the heart of the plant decays, forming a soft yellowish-brown rot which extends down into the heart of the plant (31).

Compositæ

Zinnia elegans Jacq. Affected plants of zinnia show symptoms very similar to those produced by the type virus on many plants. They are stunted and chlorotic and the flowers are of an abnormal shape. The petals tend to roll longitudinally to form a tube.

Solanaceæ

Solanum tuberosum. Potato, vars. Bliss Triumph and White Rose. The most pronounced symptom on potato plants affected with *Callistephus Virus 1 A* is the production of slender purple sprouts and purple sessile aerial tubers which develop at the end of the sprouts. Frequently dwarfed leaves develop on the aerial tubers. The margins of the leaves are rolled inwards, with the petioles often bent or curved downwards. The leaves and stems are brittle. In the later stages of the disease the lower leaves turn yellow and become dry. The incubation period of the virus in the potato plant varies from twenty to sixty-three days. All attempts to recover the virus from infected potato plants by means of the vector (*Cicadula divisa*) have so far failed (34).

Geographical Distribution of Callistephus Viruses 1 and 1A. The type virus is widely distributed throughout the United States of America, where it was first described by R. E. Smith in 1902 (35). It also occurs in Canada. In addition, the virus has been recorded from Bermuda (19), from Japan (10), from Budapest (32), and possibly from Berlin. A serious and widespread condition suggesting yellows is found in carrots and salsify in New Brunswick, while Hurst records *Callistephus Virus 1* as destructive in Prince Edward Island, where the virus is suspected of overwintering on *Leontodon autumnalis*.

The Californian strain of this virus (*Callistephus Virus 1A*) is known to occur in San Joaquin, Santa Clara and Monterey counties, and the present known distribution of this virus strain in California seems to be from the Sonoma to Los Angeles counties. A similar strain, capable of infecting celery, has also been recorded from Utah.

Control of Callistephus Virus 1. At the moment there seems little prospect of producing a resistant variety of aster, and control methods must be directed against the insect vector. It has been found that infection can be reduced by almost 50 per cent by the cultivation of the asters under screens, about 5 feet high. These screens may be made either of 18-mesh wire or of tobacco or cheese cloth (22 by 22 threads per inch). Such screens seem to prove an effective barrier to infestation of the aster beds by the leafhoppers.

It is advisable to grow the asters in the vicinity of cultivated crops rather than near to pasture land or uncultivated weedy places where the leafhopper is more abundant.

Host Range of Callistephus Virus 1 and Callistephus Virus 1A (Aster yellows viruses)

The following host list which is as complete as possible, has been compiled from the work of Kunkel and Severin. Plants marked with an asterisk are recorded as hosts of *Callistephus Virus 1A*.

Ranunculaceæ

- Ranunculus asiaticus*.*
- Adonis aestivalis* L.

Papaveraceæ

- Papaver nudicaule* L.
- Eschscholtzia californica* *
Cham.

Cruciferæ

- Cheiranthes Allionii* Hort.
- Malcolmia maritima* R. Br.
- Radicula sylvestris* (L.)
- Druce.
- Alyssum compactum*
procumbens.

Ficoidaceæ (Aizoaceæ)*Tetragonia expansa* Murr.**Portulacaceæ***Calandrinia grandiflora* Lindl.*Portulaca* sp.**Rosaceæ***Potentilla monspeliensis* L.**Loasaceæ***Blumenbachia Hieronymi* Urb.**Limnanthaceæ***Limnanthes Douglasii* R. Br.**Cistaceæ***Helianthemum chamaecistus*

Mill.

Datiscaceæ*Datysca cannabina* L.**Onagraceæ***Clarkia elegans* Doug.*Godetia grandiflora*.***Umbelliferæ***Ammi majus* L.*Daucus carota* L.*Didiscus caeruleus*.*D. pusillus* F. et M.*Levisticum paludapifolium*
(Lam.) Aschs.*Pastinaca sativa* L.*Anethum graveolens* L.*Pimpinella Anisum*.*Apium graveolens* *
(*Callistephus Virus 1A*
only).**Plumbaginaceæ***Armeria alpina* Willd.*Limonium Suworowii* Kuntze.**Primulaceæ***Primula elatior* Hill.*Anagallis linifolia* L.**Apocynaceæ***Vinca rosea* L.**Polemoniaceæ***Phlox drummondii* Hook.*P. paniculata* L.*Gilia densiflora* Benth.*G. tricolor* Benth.*Polemonium cæruleum* L.**Hydrophyllaceæ***Nemophila* sp.*Phacelia campanularia*.*P. congesta* Hook.*P. viscosa* Torr.*P. whitavia* Gray.**Boraginaceæ***Anchusa Barrelieri* Vilm.*A. capensis* Thunb.*Myosotis scorpioides* L.**Labiatæ***Dracocephalum Ruyschiana* L.*Physostegia virginica* Benth.*Satureia hortensis* L.*Lavandula vera*.**Solanaceæ***Browallia demissa* L.*Hyoscyamus niger* L.*Nicotiana rustica* L.*Petunia hybrida* Vilm.*Lycopersicum esculentum*.

Solanaceæ—continued

Solanum tuberosum *
*(Callistephus Virus 1A
only).*
Schizanthus sp.
Salpiglossis sp.

Scrophulariaceæ

Alonsoa warscewiczi Regel.
Collinsia bicolor Benth.
Linaria cymbalaria Mill.
L. maroccana Hook.
Maurandia lophospermum
Bailey.
M. scandens Pers.
Verbascum hybridum Hort.
Veronica peregrina L.
Mimulus luteus L.
Nemesia sp.
Calceolaria sp.

Gesneriaceæ

Didymocarpus Horsfieldii
Schinz.
Gloxinia sp.

Acanthaceæ

Thunbergia alata Bojer.

Plantaginaceæ

Plantago alpina L.
P. fuscescens Jord.
P. psyllium L.
P. major L.

Pedaliaceæ

Martynia sp.

Polygonaceæ

Fagopyrum esculentum
Moench.

Chenopodiaceæ

Spinacea oleracea L.

Amaranthaceæ

Amaranthus auroro.
A. caudatus L.

Valerianaceæ

Centranthus calcitrapa (L.)
Dufr.

Campanulaceæ

Lobelia erinus L., var.
compacta.

Compositæ

Acroclinium roseum Hook.
Ambrosia artemisiifolia L.
A. trifida L.
Anthemis tinctoria L.
Arctotis grandis Thunb.
Bellis perennis L.
Brachycome iberidifolia Benth.
Cacalia hastata L.
Calendula officinalis L.
Calliopsis.
Callistephus chinensis Nees.
Carthamus tinctorius L.
Centaurea imperialis Hort.
C. margarita Hort.
Charieis heterophylla Cass.
Chrysanthemum
cinerariifolium Bocc.
C. coronarium L.
C. frutescens L.
C. leucanthemum.
C. leucanthemum maximum.
C. maximum.
*C. segetum**
Cinerariu hybrida Hort.

Cirsium heteroleucum (L.) Scop.
Cladanthus arabicus (L.) Cass.

Compositæ—continued

Coreopsis lanceolata L.
Cosmidium.
Cosmos bipinnatus Cav.
Cousinia hystrix C. A. Mey.
Dimorphotheca aurantiacum
 D.C.
Echinops dahuricus Fisch.
Emilia flammea Cass.
Erigeron annnuus (L.) Pers.
E. canadensis L.
E. glabellus Nutt.
E. linifolius Willd.
E. speciosus (Lindl.) D.C.
Ethulia conyzoides L.
Eupatorium perfoliatum L.
E. urticæfolium Reich.
Felicia aethiopica (Brum.)
 O. Hoffm., var. *glandulosa*.
F. amelloides Voss.
Filago germanica L.
Flaveria repanda Lag.
Gaillardia aristata Pursh.
Galinsoga parviflora Cav.
Grindelia squarrosa (Pursh.)
 Dun.
Hedypnois cretica (L.) Willd.
Helenium autumnale L.
H. Biglovii Gray.
H. Hoopesii Gray.
H. nudiflorum Nutt.
Helichrysum arenarium D.C.
H. bracteatum.
Heliopsis laevis Pers.
Helipterum manglesii Muell.

Compositæ—continued

Hieracium alpinum L.
Kælpinia linearis Pall.
Lactuca sativa L.
Lagascæa mollis Cav.
Leontodon autumnalis L.
Leontopodium alpinum Cass.
Leptosyne Stillmani Gray.
Lindheimeria texana A. Gr.
Lonas inodora (L.) Gaertn.
Matricaria alba.
Mulgedium alpinum (L.)
 Less.
Parthenium integrifolium L.
Petasites albus (L.) Gaertn.
Pyrethrum sp.
Rudbeckia hirta L.
Sanvitalia procumbens Lam.
Schkuhria abrotanoides Roth.
Scolymus hispanicus L.
Spilanthes acmella (L.) Murr.
Tagetes erecta L.*
T. patula.
Taraxacum officinale Weber.
Thelesperma hybridum Voss.
Tolpis barbata Gaertn.
Tragopogon floccosus W. et K.
Tridax porrifolius L.
T. trilobata (Cav.) Hemsl.
Ursinica artemoides (L.)
 Benth. et Hook.
Zacyntha verrucosa Gaertn.
Zinnia elegans * (*Callistephus*
 Virus 1A only).
Z. multiflora L.

LACTUCA VIRUS 1. Jagger

Synonym. *Lettuce Mosaic Disease Virus*, Jagger, 1921.

The Virus and its Transmission. There is very little information concerning this virus. Jagger, in America, considers that the insect vector is the aphid *Myzus persicæ* Sulz., while there is some

evidence that the vector in England is *Macrosiphum sonchi*. The virus is sap-transmissible and is also apparently seed-transmitted, since it has been shown that of 700 plants grown from mosaic seed 37 per cent were affected with the disease (20).

Disease caused by *Lactuca Virus 1*

Compositæ

Lactuca sativa. Lettuce. *Mosaic* (see Fig. 34, E). The first symptom of the disease in Romaine lettuce (var. Paris White Cos) is a yellowish discolouration along the smaller veins of the younger expanding leaves. This symptom is usually evident for only a few days, giving way to a general yellowish, discoloured appearance of the whole plant. All gradations of this discolouration occur. Close examination usually reveals irregular blotches of an approximately normal green colour, mostly situated along the larger leaf-veins. The blotching varies from a few barely perceptible green areas on a yellowish leaf to numerous pronounced green spots, giving a marked mottled appearance to an occasional plant. The leaves of diseased plants are generally rather more wrinkled than those of normal plants.

Diseased plants make a stunted growth. In severe cases the plants are decidedly undersized and occasionally the leaves form only a rosette, with no indications of a folding together of the tips to form a head (13).

Geographical Distribution. Lettuce mosaic has been recorded from several localities in the United States. It is also very prevalent in certain parts of England, especially in the Evesham and Cambridge districts, where it seems to be increasing in abundance.

Control. If it is proved that *Lactuca Virus 1* is carried in the seed it is important to obtain seed from certified virus-free stocks. On small-scale plantings it may be possible to restrict the spread of the disease by the use of insecticides to kill the aphid vector and by the eradication of diseased plants.

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CHAPTER IV

Nicotiana Viruses 1, and 1A-1D, 2-12 and 12A-12B.

NICOTIANA VIRUS 1. (Mayer) Allard

Synonyms. *Mosaikkrankheit des Tabaks*, Mayer, 1886; *Tobacco Pockenkrankheit Virus*, Iwanowski, 1890; *Tobacco Mosaic Virus*, Allard, 1914; *Tobacco Calico Virus*, Clinton, 1903; *Tomato Mosaic Virus*, Clinton, 1909; *Ordinary Tobacco Mosaic Virus*, Johnson, 1926; *Tobacco Green Mosaic Virus*, McKinney, 1926; *Tobacco Mosaic A Virus*, Fernald, 1925; *Tobacco True Mosaic Virus*, Valleau and Johnson, 1927; *Tobacco Severe Mosaic Type 1 Virus*, Johnson, E. M., 1930; *Tobacco Distorting Mosaic Virus*, Duggar and B. Johnson, 1933; *Ordinary Field Type Tobacco Mosaic Virus*, Kunkel, 1934.

The Virus

In consequence of its infectious nature, its power of resistance and its general suitability for experimental work, the virus of tobacco mosaic has been studied to a greater extent than any other plant virus. There is, therefore, a considerable amount of information available upon its physical properties and other characteristics.

Reactions with Various Chemicals. The effect of miscellaneous chemicals upon the infectivity of the virus has been studied (5). Stanley (90) found that with the exception of brucine the only reagents that affect this virus directly are oxidising agents, protein-precipitating agents, and materials causing a hydrogen-ion concentration known to inactivate the virus. The fact that tobacco mosaic virus was found to be unaffected over long periods of time by concentrations of mercuric chloride known to be germicidal in action is of interest. Virus in purified preparations was affected by mercuric chloride at pH 6, 7 and 8 to a greater extent than at pH 3, 4 and 5. These results are thought to be in accord with available evidence that the virus is protein in nature.

Charcoal may cause no change, an increase or a decrease in the infectivity of tobacco mosaic virus depending upon the ratio

of amount of charcoal to amount of virus. The virus may be completely removed from solution by adsorption on charcoal, the maximum adsorption occurring at pH 3 to 5 with finely divided charcoal. Oil of mustard (2 per cent), digitalin (5 per cent), nicotine (1 to 2 per cent) and atropin (1 to 2 per cent) inactivate the virus after various periods, while saponin (2 per cent) has no appreciable effect on infectivity (24). Inhibition of tobacco mosaic virus infection by tannic acid appears to result in part from an interaction of virus and acid, since complete inhibition depends upon concentration of acid and time of action. A part of the effect may also result from the action of tannic acid on the host plant, and it is not certain whether the inhibition of infection results from a change in the virus, in the protoplasm of the host, or both (100).

The reduced form of ascorbic acid in concentrations as low as 0.03 mg. per cubic centimetre can produce complete inactivation of purified preparations of the virus. Inactivation takes place only when the ascorbic acid in the virus suspension undergoes oxidation by atmospheric oxygen (55).

Reactions with Enzymes. Tobacco mosaic virus is not inactivated by trypsin, but this enzyme does cause an immediate loss of infectivity of about 60 per cent. Stanley (88) suggests that this immediate loss is due to an effect on the reaction of the host plant to the virus rather than to any proteolytic action of the enzyme on the virus. Bawden and Pirie (8) offer the alternative suggestion that trypsin may form a loose complex with the virus.

Various workers have claimed that pepsin has no effect on tobacco mosaic virus, whilst Stanley (89) considers that crystalline pepsin slowly inactivates it, complete inactivation being obtained only after several days' incubation at pH 3 and 37° C. in the presence of 0.17 per cent pepsin. According to Lojkin and Vinson (56) papain inactivates the virus in neutral phosphate solution, while an crepsin preparation is effective in reducing the infectivity of the virus only after an incubation period of several days. No enzyme, however, has yet been found which will attack the crystalline virus protein described on p. 233.

Serological Reactions. Tobacco mosaic virus which has been subjected to purification by the use of lead acetate, acetone, trypsin, aluminium hydroxide, barium hydroxide, alternate freezing and thawing, and silver nitrate precipitation retains its ability to react specifically with tobacco mosaic immune serum prepared either from whole sap or from purified tobacco mosaic juice.

Moreover, the ratio, amount of virus to precipitinogenic power, remains constant throughout such purification (19). Virus which has been inactivated by nitrous acid retains its power to flocculate antiserum (91A).

Thermal Death-point. The virus in undiluted juice of mosaic-diseased tobacco plants is inactivated in one minute at 96° C., in ten minutes at 98° C., in eighty minutes at 90° C., in thirty-two hours at 85° C., in twelve days at 80° C., and in forty days at 75° C. It is not completely inactivated when held for seventy days at 68° C. Virus in juice of mosaic-diseased tobacco plants, diluted 1 : 20 with water, is destroyed in one minute at 92° C., in ten minutes at 88° C., in seventy minutes at 85° C., in thirteen hours at 80° C., in seventy-two hours at 75° C., and in twenty days at 68° C. (69). The virus is not entirely inactivated by ten minutes' exposure to 100° C. dry heat.

Dilution End-point. While the tolerance to dilution exhibited by a virus must obviously depend on its initial concentration, and while this can be influenced by many factors, the virus of tobacco mosaic withstands a very high degree of dilution as compared with the dilution end-points of other viruses under comparable conditions. Extracts of tobacco mosaic virus from a series of different hosts give good infections up to dilutions of 1 : 1,000,000 and probably higher. The species of host plant from which the virus is taken appears to have no bearing on the virus concentration with the possible exception of *Solanum nigrum* (49).

Longevity *in vitro*. In sterile filtered juice the virus retains its infectivity for a long period, probably several years, but in ordinary extracted sap the resistance to ageing is not so great. Virus sap stored at room temperature becomes reduced to 2 per cent of its original strength in a month, while similar sap stored in the frozen condition retains about 15 per cent of its strength as long as one and a half months (88).

Desiccation. Tobacco mosaic virus can be precipitated with alcohol and completely dried over sulphuric acid. In this dried state and in desiccated leaf-tissue the virus retains infectivity for a period of a year or more.

Particle-size. The average particle-diameter of this virus has been measured by several workers. By the method of ultra-filtration through Elford's gradocol membranes the size has been calculated to be 15 m μ (57) and 11 m μ (101). By centrifugation the particle-size is found to be much greater, *i.e.*, 50 m μ (11).

. A Crystalline Protein Intimately Associated with Nicotiana

Virus 1. Stanley (91) has isolated a crystalline protein, which has the properties of tobacco mosaic virus, from an extract of Turkish tobacco plants infected with this virus. The extract was prepared by grinding frozen plants, adding disodium phosphate, and pressing out the liquid. The press cake was extracted a second time with dilute disodium phosphate, and the two extracts were filtered through celite and combined. The virus protein was obtained from these extracts by precipitation with ammonium sulphate. The virus protein was reprecipitated with ammonium sulphate several times, with loss of much colour, and most of the remaining colour was then removed with lead subacetate. The virus was adsorbed on, and removed from, celite several times and then crystallised in the form of small needles, about 0·02 mm. long, by the addition of a solution of 5 per cent glacial acetic acid in 0·5 saturated ammonium sulphate.

By the use of a simplified method of purification and by carrying the process further it has been shown recently that this protein can be obtained in liquid crystalline states. The protein, when precipitated with acid and dried, has the following analytical figure: C, 51 per cent; H, 7·1 per cent; N, 16·7 per cent. The sulphur content varies from 0·2 to 0·7 per cent, and there is 0·5 per cent phosphorus and 2·5 per cent carbohydrate. Highly purified protein solutions, if stronger than 2 per cent, separate into two layers on standing. The lower layer, which may be water clear, is liquid crystalline. The upper layer shows, on gentle agitation, the phenomenon of anisotropy of flow. This has previously been noticed by Takahashi in clarified sap (99). This phenomenon of anisotropy of flow, together with the information derived from X-ray photographs of the crystals, suggests that the molecules of this protein may be rod-shaped. The conclusion that these rods are in fact virus particles seems to be reasonable, but it is not yet proved, nor is there yet any evidence that these particles exist as such in infected sap. It is interesting to find that the two strains of *Nicotiana Virus 1*, i.e., 1A and 1C, yield a crystalline protein in each case similar to that of the type virus. These three proteins show no gross physical or chemical differences and are serologically related (9).

Effect of Ultra-violet Light. The virus of tobacco mosaic is rather resistant to the action of ultra-violet light at 0° C., and is also more resistant than the vegetative stages and spore forms of certain bacteria. The resistance ratio of virus to bacteria is thought to be about 200 : 1 (21). Price and Gowen (71A) find

that the survival values of the virus exposed to ultra-violet light follow a simple exponential curve.

Effect of X-rays. The survival ratios for virus exposed to X-rays follow a simple exponential curve which suggests that the absorption of a single unit of energy in a virus particle is sufficient to cause inactivation of the particle (26A).

Electrophoresis. According to Takahashi and Rawlins (98), unpurified tobacco mosaic virus migrates to the anode (positive pole) during electrophoresis between pH 4 and 9. No migration of the virus was detected between pH 3 and 1·2.

Methods of Transmission. The virus is sap-transmissible and is one of the most infectious of the plant viruses, the breaking of a trichome with a contaminated instrument being sufficient to transmit the virus. Tobacco plants can also be infected artificially with this virus by spraying a suspension on to the leaves by means of an atomiser. It is not quite clear by what mechanism this type of infection is achieved, but it is suggested that the atomised particles of virus suspension may be either directly shot into fully open stomata or that they enter the stomata during the spreading of the film formed by the atomiser. The mechanism of this supposed penetration of the virus particle into the living cell from the sub-stomatal cavity is not yet understood (22). On the other hand, it may be that the virus enters through broken hairs or minute wounds on the leaf surface.

Owing to the resistance of this virus to the effects of drying and to its fairly high thermal death-point, it frequently retains its infectivity in cigarettes and pipe tobacco. These are therefore potential sources of virus infection to susceptible crops, the virus being carried to the plants on the hands of the workers. Similarly, infection is liable to be spread by means of chewing tobacco (25, 105). A certain amount of spread of the virus may also take place in the field by contact with infective material remaining in the soil, but this source of infection is probably negligible, especially as the virus apparently cannot infect tobacco plants through the roots. The virus is, of course, graft transmissible.

Insect Vectors. It is a curious and interesting fact that so infectious a virus as that of tobacco mosaic should yet have so slight a relationship with insect vectors. The aphides which occur commonly on the tobacco plant are apparently unable to transmit the virus, and, indeed, the vector under field conditions, if such exists, is not known. Hoggan (86) states that the aphid *Myzus*

persicae is unable to transmit tobacco mosaic virus to and from the tobacco plant, and this insect, apparently, cannot transmit the virus at all. On the other hand, the aphides *Myzus pseudosolani* (p. 542), *Myzus circumflexus* (p. 537), *Macrosiphum gei* (= *solanifolii*) (p. 532) will apparently transmit the tobacco mosaic virus from tomato to tobacco, but not from tobacco (37).

Seed Transmission. The question of the seed transmission of tobacco mosaic virus is a vexed one, and a definite answer cannot yet be given. At the plant virus research station at Cambridge, where tobacco plants have been grown from seed in large numbers for many years past, infections occur which can hardly be explained except on the theory of seed transmission. Small tobacco plants in the four-leaf stage, growing under insect-proof conditions, may occasionally be found infected with tobacco mosaic, and this is frequently of a type or strain different from any under study at the time. Bewley (14) is of the opinion that the virus is seed-borne in a small percentage in the case of tomato plants, while Berkeley and Madden are of the same opinion (13). It has been shown that the virus is present in the pollen grains of infected tobacco plants, though it does not necessarily follow that transmission of the virus accompanies fertilisation (35). On the other hand, Gratia and Manil (29) claim that the virus is absent from the pollen grains and anthers. They consider that the virus gradually becomes attenuated and ultimately disappears during the differentiation of the floral organs. A paper just published by Doolittle and Beecher (*Phytopath.*, 1937) offers evidence of the seed-transmission of this virus through the planting of *freshly extracted* seed of tomato.

Differential Hosts

The most common strain of tobacco mosaic gives local lesions only on the inoculated leaves of *Nicotiana glutinosa*, more lesions developing at high than at low temperatures (see Fig. 37, B). Under normal conditions there is no systemic spread of the virus from the point of inoculation, but if the temperature is very high, above 90° F., there may be invasion of the young leaves, which become highly necrotic. On tobacco, *N. tabacum*, this virus does not as a rule and under normal glasshouse conditions give local lesions and invariably becomes systemic. It must be pointed out, however, that there are some strains of the virus which do give local lesions on tobacco, and these may or may not be followed by systemic infection. Some varieties of the French or snap bean

(*Phaseolus vulgaris*), such as the American Golden Cluster, react to inoculation, with the formation of small reddish local lesions, without further spread of the virus. *Physalis viscosa* appears to be completely immune (39).

Diseases caused by Nicotiana Virus 1

Ranunculaceæ

Delphinium consolida. Larkspur. Infected plants are stunted in growth compared with normal plants, and there is considerable chlorosis. The leaves are mottled, somewhat malformed and show necrotic lesions. The disease is systemic. Intracellular inclusions (X-bodies) and striate material are present in the chlorotic tissue of larkspur affected with this virus (28).

Polygonaceæ

Fagopyrum esculentum. Buckwheat. The leaves exhibit numerous pale chlorotic spots, together with a certain amount of malformation which distorts the leaf outline. There may be some necrosis and the disease is usually systemic. It is not certain whether intracellular inclusions are present, but they have not been observed (28).

Chenopodiaceæ

Spinacia oleracea L. Spinach. Infected spinach plants show yellowing and mottling of the leaves. Some stunting and necrosis may develop, but these are much less severe than in the disease caused by *Cucumis Virus 1* (cucumber mosaic virus) (see p. 56). Intracellular bodies are present in the diseased leaves, but these are small and few in number (28). The disease is systemic. In the spinach variety Bloomsdale Savoy, sap-inoculation gives 50 to 90 per cent infection after sixteen to twenty days' incubation. The first symptom is a mottling of the older foliage with slightly raised dark green areas in the paler green tissue. At a more advanced stage the leaves become increasingly puckered and general dwarfing is observed. Yellowing of the older foliage is followed by death a month or six weeks after inoculation (50).

Beta vulgaris. Garden beet. In garden beet the virus does not spread from the inoculated leaves which become yellow and necrotic and usually fall off. The same is true of the action of this virus on sugar beet.

Ficoidaceæ

Tetragonia expansa. New Zealand spinach. Local infections only result on New Zealand spinach, the inoculated leaves become chlorotic with some necrosis and later drop off.

Leguminosæ

Phaseolus vulgaris. French or snap bean. Varieties, Early Golden Cluster and Green Pod Refugee. *Nicotiana Virus 1* produces local lesions only when rubbed on to the leaves of the above varieties of *P. vulgaris*. These lesions appear two days after inoculation and are about $\frac{1}{2}$ mm. in diameter. They consist of a pale necrotic area surrounded by a ring of dark red tissue, they may increase slightly in size, but are seldom larger than 1 mm. in diameter (68).

Compositæ

Emilia sagittata. Tassel flower. Infection of this plant is erratic and the symptoms are very slight, though mottling and chlorosis can be induced and partial defoliation. In the Cape Marigold, *Dimorphotheca aurantiaca*, the symptoms consist of necrosis of the mid-ribs of the leaves with subsequent curling and dwarfing of the plant. The disease produced in *Zinnia elegans* is variable, it may be localised, causing only local lesions, or it may spread to other leaves, resulting in the appearance of small round yellow spots. These spots may remain yellow for a few days and finally disappear or coalesce to form a large yellow blotch. In the French marigold (*Tagetes patula*) the disease is similarly erratic, but when the virus becomes systemic the affected plant is frequently stunted and malformed. Occasionally the disease takes the form of scattered chlorotic or necrotic spots on the leaves (28).

Polemoniaceæ

Phlox drummondii. Only local symptoms develop on phlox, and they take the form of mottling of the inoculated leaf and some yellowing.

Hydrophyllaceæ

Phacelia whitlavia. California bluebell. In this plant systemic infection develops, producing a mottling and deforming disease very similar to that produced in the tobacco plant. Leaves are distorted, the outline is irregular and broad bands of green develop in the tissue along the veins. X-bodies are numerous in

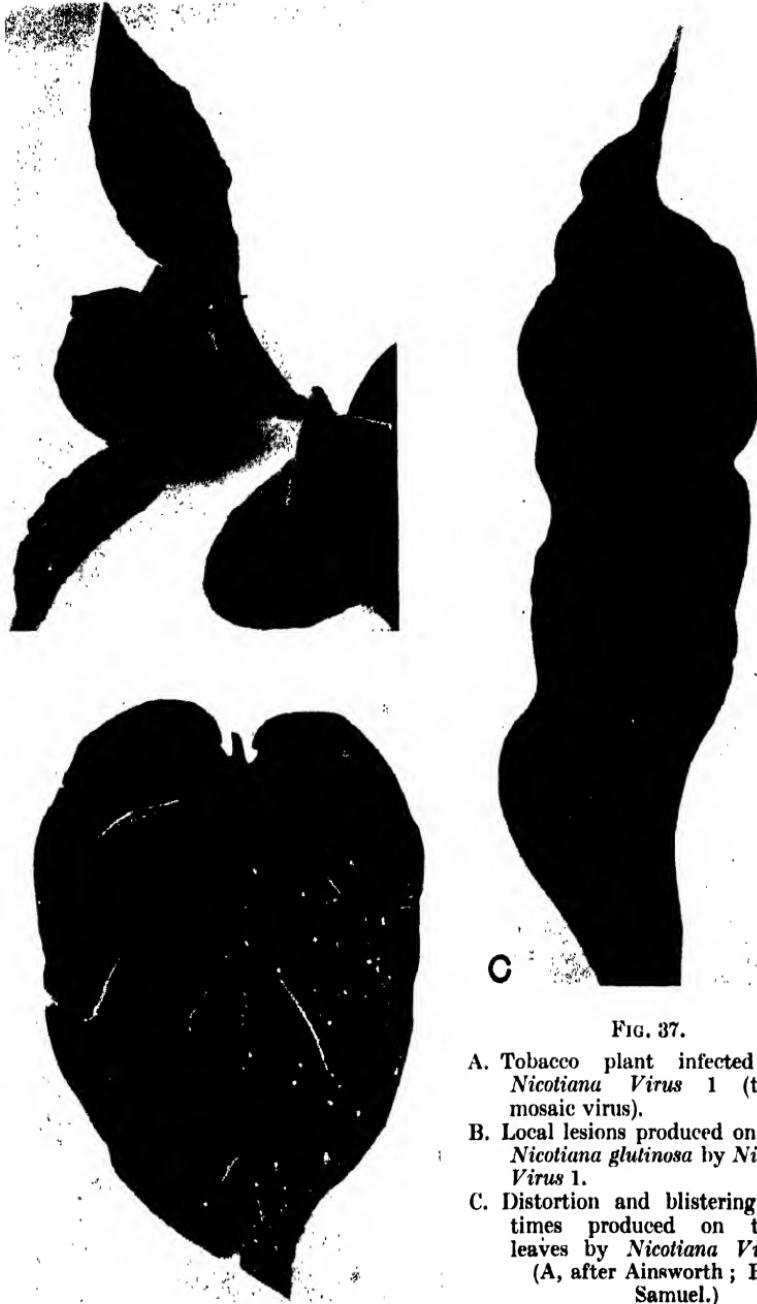


FIG. 37.
A. Tobacco plant infected with *Nicotiana Virus 1* (tobacco mosaic virus).
B. Local lesions produced on leaf of *Nicotiana glutinosa* by *Nicotiana Virus 1*.
C. Distortion and blistering sometimes produced on tobacco leaves by *Nicotiana Virus 1*.
(A, after Ainsworth; B, after Samuel.)

the chlorotic tissue, and there is usually some striate material present. Other species of *Phacelia* show similar reactions (28).

Boraginaceæ

Cynoglossum amabile. Chinese forget-me-not. A systemic disease is produced in this species with mottling of the leaves and a certain amount of malformation and stunting.

Solanaceæ

It is in connection with the Solanaceæ, and particularly the tobacco and tomato plants, that this virus assumes its greatest importance. A very large number of plants belonging to this family are susceptible to infection with tobacco mosaic virus, and these are dealt with as fully as possible.

Nicotiana tabacum. Tobacco. *Tobacco mosaic* (see Fig. 37, A and C). As a rule there are no local lesions formed on the inoculated leaf, but under certain environmental conditions these do develop. When the temperature and light intensity are high, small circular, faintly chlorotic spots may appear on the rubbed leaves. Yellowish primary lesions appear on the rubbed leaves of Turkish tobacco plants during the summer. First signs of systemic infection are the clearing of the veins of the youngest leaf, followed by mottling, frequently accompanied by some form of distortion. The youngest leaf may develop with the outer edge slightly turned upwards, so that a rim is formed round the leaf. Following upon this, large blisters of green tissue and raised or sunken yellow areas may develop, together with a marked mottling of dark and light green and considerable malformation and distortion of the leaves. In some cases the lamina of the leaf is so reduced that a filiform or "shoe string" effect is produced. Frequently yellow patches develop on the leaves due to the presence in the plant of more than one virus strain.

Under high temperature conditions and great humidity the disease takes on a more necrotic and destructive form and numerous brown lesions or patches develop on the leaves. The clinical picture of tobacco mosaic is greatly influenced by environmental conditions. At temperatures above 98° F. the symptoms may disappear entirely; this is known as the "masked" condition. Similarly, at temperatures below 51° C. masking of the symptoms also occurs (27). Partial sterility of tobacco may be caused by this virus, 48 to 50 per cent of the pollen being abortive (53).

Histopathology. As regards the histology of this disease in the tobacco plant, cell-inclusions of two types are generally present in the chlorotic areas of mottled leaves. These are the X-bodies and the striate material. The X-bodies are numerous, they are highly vacuolate and are usually associated with the cell nucleus. They may also be found in the pollen grains from infected tobacco plants (40).

It has recently been suggested that the X-bodies and striate material may be the same as, or related to, the crystalline virus protein referred to on p. 233. Beale (10) found that by acidification of the cell contents, intracellular precipitation of needle-shaped crystals was obtained. These crystals bore a marked resemblance to the crystalline virus protein.

Other species of *Nicotiana* which respond similarly to this virus are as follows: *Nicotiana sylvestris* Spegaz and Comes, *N. longiflora* Cav., *N. rusbyi* Britton, *N. suaveolens* Lehm, and *N. palmeri* A. Gray. The symptoms on *N. trigonophylla* Dun. are of the same general type, but mottling and distortion are less pronounced.

Nicotiana glutinosa (see Fig. 37, B). The reaction of this plant to the virus is unusual in that local lesions only develop and no systemic spread of the virus follows except under high temperature conditions. Primary lesions develop on the inoculated leaf in the form of a necrotic spot, first appearing as a tiny dark sunken area which, upon drying, becomes light tan in colour. On the fourth day after inoculation, at temperatures between 20° and 25° C., a ring of dark brown pigment develops on the periphery of the lesion. This ring makes the dead spot conspicuous and allows it to be differentiated from other nearby lesions (89).

Nicotiana rustica. In young plants of *N. rustica* from two to five days after infection, starch retention lesions (see p. 241) can be demonstrated in inoculated leaves. About the fifth day after inoculation necrotic primary lesions appear, followed soon by a clearing of the veins somewhat similar to that shown by *N. tabacum*. The youngest leaves are retarded in their development, begin to pucker, and appear abnormally yellowish in colour. The veins of developing leaves are often affected by necrosis, and the youngest leaves die soon after the virus reaches them. In general, the attempt to put out new leaves is followed by recurring necrosis of so severe a kind as to culminate eventually in the death of the plant. In some cases buds at the base of the stem continue to develop green leaves for many days, but these also are finally

invaded. As a rule plants which are infected while young succumb to the disease (89).

Nicotiana glauca. When inoculated as a young plant, *N. glauca* shows a faint but typical mottling pattern. As growth continues, however, this mottling may disappear entirely from the plant, which becomes a symptomless carrier. Secondary lesions in *N. glauca* in the form of separate spots can be demonstrated by staining starch retention patterns with iodine.

Nicotiana paniculata L. *N. tomentosa* Ruiz and Pav. The reaction of these two species to the tobacco mosaic virus is interesting and unusual. In addition to the mottling and distortion characteristic of the virus upon the tobacco plant, protuberances, or "enations," arise at the edge of the chlorotic areas in the leaf. These outgrowths are leaf-like in appearance, extending downwards from the lower surface of the leaf. They vary considerably in size, the larger ones sometimes extending downwards as much as 1 inch from the leaf surface. The smaller ones are often so small that they can only be seen when the leaves bearing them are viewed by transmitted light, in which case they appear as dark green lines. Some of these enations have the appearance of small inverted cups. Others appear as double leaf-like protrusions emerging from the surface of the leaf. The outgrowths are morphologically and structurally similar to the leaves from which they develop. The inner surface of the outgrowth, which faces the chlorotic area around which the protrusion develops, is dark green in colour like the upper side of a normal leaf. The outer surface which faces away from the chlorotic area is greyish like the under side of a normal leaf. Enations only appear on leaves developing after the appearance of systemic symptoms and never on inoculated leaves. They usually appear on plants about twenty days after inoculation. Histological examination of the outgrowth shows that they arise through cell division and enlargement from the lower cell layers of the leaf. Palisade tissue has been found, in a number of instances, to develop in the lower cell layers of the leaf between the two sides of outgrowths. Large outgrowths, in cross-section, consist of seven cell layers extending downwards from the lower surface of the leaf. These cell layers are differentiated into epidermis, palisade and spongy parenchyma (42).

Nicotiana multivalvis. A very severe and persistent yellowing characterises infected plants of *N. multivalvis*. Inoculation of young plants in the rosette stage is followed after a few days by

clearing of the veins, with subsequent yellowing of all recently formed leaves. Persistent reduction of the growth rate of the plant occurs ; in this stage of the infection the plants often die. After an extended period of yellowing and stunting, which may last from one to five weeks, renewed growth results in the formation of mottled and distorted leaves or of narrow leaves in which yellowed tissue fails to develop and only the green areas expand to appreciable width of lamina. Plants in this condition may eventually die in the rosette stage. Blossom buds in older infected plants fail to open, and close examination shows the white tissues of the blossom buds to be discoloured, and abscission occurs sometimes at a touch. *N. quadrivalvis* and *N. clevelandii* behave similarly, though the latter is less intensely yellowed (39). Seed from such infected plants gives rise to healthy seedlings.

Datura Stramonium. Thorn apple, Jimson weed. Local necrotic lesions develop on the inoculated leaves. These lesions are somewhat similar to those produced on *N. glutinosa*, being lighter in the centre than at the periphery. Holmes (38) considers that systemic infection does not result, but under the prevailing conditions at Cambridge, *Datura* plants are almost invariably killed by a rapid necrosis of the petioles and stem. Plants so affected fall over to one side and die very quickly.

Solanum melongena, var. Black Beauty. Egg plant. Young plants of this species are often killed by the systemic spread of tobacco mosaic virus. Necrotic spots and streaks appear along the veins in developing leaves some days after the appearance of primary lesions on the inoculated leaf, and portions of stem near the top of the plant are often involved. Some defoliation may take place, leaves with secondary lesions dropping off and leaving the stem with only the smallest leaves at the top. When plants have been affected by the systemic disease for some time, stem lesions and dead streaks in petioles and mid-veins may be numerous and conspicuous. In cross-section the dead tissues are seen to be superficial in some cases and to lie more deeply in others. Plants infected when large and nearly mature do not develop systemic symptoms, and the increase with age of resistance to systemic infection is conspicuous in this plant (39).

S. melongena, var. Hangchow Long. This variety of egg plant is a good example of a symptomless virus carrier. Inoculation of the leaves of this plant results in the production of a large amount of virus locally without affecting either the appearance of these leaves or, subsequently, the rest of the plant. Other

varieties of egg plant behaving similarly are *S. melongena*, var. *creviviolacea* and *S. melongena*, var. *Kuli*. Some varieties, however, such as Long White and Peking Green, show mottling at the time of infection, later becoming symptomless carriers.

Solanum nigrum. Black nightshade. This plant shows only slight distortion, while the leaves are mottled, though the mottling is not very pronounced.

Physalis angulata. Holmes (39) has shown that the genus *Physalis* contains species which respond in many different ways to infection with tobacco mosaic virus. *P. angulata* develops small necrotic primary lesions on its inoculated leaves about three days after inoculation. The lesions break down more completely on the older leaves, appearing imperfectly as traces of necrosis. On younger leaves the lesions are sometimes solid necrotic spots, and sometimes delicate rings or double concentric necrotic rings with green tissue between the rings of necrosis. Defoliation follows the appearance of the primary lesions, all inoculated leaves being lost within a week or two of the time of inoculation. Death usually ensues for a systemic necrosis, but occasionally the virus is lost through abscission of the inoculated leaves and no further infection can take place.

P. peruviana. Leaves near the top of an infected plant of this species may show slight clearing of the veins some days after inoculation. Later-formed leaves display an obscure mottling with small green blisters on a slightly lighter yellowish-green background. Considerable defoliation may occur, but chiefly in the partly expanded leaves affected with the systemic disease. The inoculated leaves remain attached to the plant.

P. alkekengi. No symptoms develop in *P. alkekengi*, and the plant acts as a symptomless carrier of the virus (65).

P. viscosa. This species appears to be immune from infection with the tobacco mosaic virus. All attempts to infect it by inoculation or grafting have given negative results (39).

Hyoscyamus niger L. The symptoms of infection are somewhat similar to those on tobacco. The plants show clearing of the veins, and this is followed by severe stunting and yellowing. Later leaves with a pronounced or brilliant mottling are produced.

Petunia spp. Garden varieties. Petunias on the whole give a clinical picture very similar to that of mosaic tobacco with pronounced mottling and distortion of the leaves. In some varieties the margins of the youngest leaves become upwardly curved, giving a spoon- or cup-shaped appearance.

Nicandra physaloides. The reaction of this species to the virus is somewhat similar to that of the symptomless carriers in allowing only erratic spread of a small amount of virus to the partly developed leaves at the top and producing a few discrete secondary lesions on medium-sized and large leaves. It differs from the symptomless carriers, however, in allowing the chlorophyll content to be affected where there is systemic spread of the virus with the production of a yellow spotting systemic disease. Systemic symptoms also appear in the flowers of *N. physalodes* where there is an increase in the intensity of the blue-violet colour of the corolla. The flowers are somewhat distorted.

Lycium ferocissimum. In this plant yellowish primary lesions appear on the inoculated leaf, and in the systemic infection yellow spots become visible on leaves just below the youngest, most rapidly expanding leaves. The affected tissues do not expand as rapidly as the green tissues in this plant, and the affected leaves roll their margins upward.

Capsicum frutescens. Pepper. Certain varieties of pepper, notably Anaheim Chili, Tabasco and Creole, show some systemic necrosis. A characteristic symptom is the abnormal position of the inoculated leaf a few days after infection. The petiole bends backwards so that the leaf, instead of pointing outward and slightly upward, points straight down. Abscission of flowers and young fruits occurs soon after this. A severe and persistent yellowing is a characteristic of infection in some varieties.

Lycopersicum pimpinellifolium Mill. Red currant tomato. Soon after infection of young plants an obscure but typical mottling of young leaves is noticed. After a time this mottling disappears and the plant becomes a carrier. Infected and uninfected plants produce leaves and branches of the same size, shape and position on the plant, and bear equally numerous fruits, which ripen simultaneously.

L. esculentum. Tomato. The symptoms of this very common disease known as "ordinary" or "mild" tomato mosaic are well known to most growers of the tomato. Affected plants exhibit a mottling with raised dark green areas and some distortion of the youngest leaves (see Fig. 47, A). Under conditions of high temperature and high light intensity the mottling is frequently severe, but stunting is slight. During the winter or under conditions of low temperature and particularly low light intensity the mottling is imperceptible, but stunting and leaf distortion are severe with development of "fern leaf" and production of

anthocyanin in the stem. There is no necrosis of stem or leaves and the fruit is normal in appearance.

It has been shown that there is a high negative correlation between the growth rate of seedling tomato plants and the incubation period of the virus in the plant (4). The effect of this virus on the health of the tomato plants is not very severe. It has been calculated that mosaic reduces the yield of glasshouse-grown tomato plants by about 8 per cent (34). Different soil conditions do not appear to influence very greatly the character of the disease.

The question of the seed transmission of the virus is an important one, and opinion on this question seems to be divided. Bewley and Corbett (15) and Berkeley and Madden (13) offer evidence that seeds collected from mosaic-diseased tomato plants give rise to diseased plants. On the other hand, it is certainly true that large numbers of seeds collected from mosaic-diseased plants give rise to normal plants. It is, of course, possible that a percentage of seedlings may become infected by a mechanical contamination of the developing seed with virus which is adhering to the seed coat. Since the virus is present in the ripe fruits of infected plants it is clear that it must also be in contact with the outside of the seeds (see also p. 235).

Gesneriaceæ

A species of *Gesneria* showing symptoms of a severe disease was found to be infected with a strain of tobacco mosaic virus. On tobacco this strain produced a very faint mottling, much less pronounced than the usual symptoms caused by this virus. Nevertheless, several tests indicated that the virus was actually a strain of tobacco mosaic virus (*Nicotiana Virus 1*).

On *Gesneria* the disease consisted of numerous necrotic spots, sometimes in the form of rings accompanied by a general chlorosis of the whole plant and stoppage of growth. On inoculation to *Gesneria* numerous small lesions of a red colour developed on the rubbed leaves.

Pedaliaceæ (= Martyniaceæ)

Martynia louisiana Mill. Unicorn plant. This plant was the first species, outside the Solanaceæ, shown to be susceptible to *Nicotiana Virus 1*. Symptoms take the form of mottling and malformation of the leaves and stunting of the whole plant.

Distribution. The distribution of tobacco mosaic virus seems practically world-wide; it is found wherever the tobacco plant is grown, and has been carried, probably in smoking tobacco, to other countries, where it annually causes disease in the tomato crop.

Control Measures. Since the virus of tobacco mosaic is not apparently insect-borne, or only so transmitted with difficulty, measures for its eradication consist largely in attention to plant hygiene and careful husbandry. These measures will be discussed in relation to the tobacco and tomato plants, the two crops most commonly affected by this virus. In considering the question of control, it must be remembered, firstly, that the virus is extremely infectious, and, secondly, that the chief agent in its dissemination is man himself. The following are some important recommendations for the control of the disease in the tobacco plant. All plants in the seed bed showing a suspicious mottling should be removed and burned, together with their immediate neighbours. When the plants have become established in the field, a careful examination for mosaic should be made before pruning operations are commenced and all diseased plants should be removed. After handling any tobacco, cured or otherwise, the hands should be thoroughly washed with soap and water before working on the seed beds. No tobacco trash of any kind should be used on the bed, either for burning or fertilising, as stalks and pieces of leaf-tissue if left about in the soil may cause infection. It should also be remembered that pipe tobacco, cigarettes and chewing tobacco are all potential sources of virus infection. Remove so far as practicable weedy patches from the vicinity of the beds, especially solanaceous weeds, as these may harbour the virus. A variety of tobacco resistant to the mosaic virus has recently been described (66). The variety is known as *Ambalema*, and although it may become infected with the virus, the symptoms produced are very slight and transitory, finally disappearing altogether.

Many of the foregoing recommendations also apply to the disease in the tomato plant. Here again it is only necessary for one or two plants to be diseased for the virus to be carried by the worker to every other plant in the crop. Particular attention should be paid to the young potted plants or, if it is a field crop, to the seed bed. Any young plants showing a mottling of the leaves should be destroyed. Only seed from mosaic-free plants should be used. Washing the hands with soap and water frequently and periodic sterilisation of the pruning knife during

cultural operations will materially reduce the spread of the virus (58, 105).

Strains of Nicotiana Virus 1

Recent work has shown that a number of plant viruses and particularly the virus of tobacco mosaic occur as several strains of the virus in question, and such strains while having the general properties of the type virus may yet produce entirely different disease symptoms. Jensen has shown that a large number of yellow and other strains of tobacco mosaic may be isolated from an initial green strain of this virus, and he presents some experimental evidence suggesting that such strains arise by what, for want of a better word, may be called "mutation" during the process of inoculation (41).

It is not practicable or advisable at present to attempt to describe all the different strains of the tobacco mosaic virus, and attention will therefore be confined to two or three important viruses which appear to be strains or variants of *Nicotiana Virus 1*. Since the general properties of these virus strains are similar to the type virus already described, only the diseases produced by them will be dealt with.

NICOTIANA VIRUS 1A. Ainsworth and Smith

Synonym. *Tobacco Distorting or Enation Virus*.

The chief importance of this virus lies in its effect upon the tomato plant, in which it produces a severe and characteristic disease (see Fig. 38). The initial symptoms on young tomato plants consist of a mottling of the young leaves, quickly followed by the distortion which is so characteristic of this infection. The malformations of the leaves are of several quite distinct types. There is, firstly, the complete suppression of the lamina so that the leaves consist simply of long thin threads, or, alternatively, there may be leaves with half laminae only. Secondly, there may occur large numbers of very small leaflets, placed close together, forming a curious kind of "fern leaf" (see also p. 69) and ending in a corkscrew tendril. The tips of individual leaflets may be prolonged, giving such leaves a curious spidery appearance. A third effect is the production, on the under surfaces of the leaves, of outgrowths (*enations*) or small additional leaves (see Fig. 39, B). Added to these abnormalities there is a tendency to overgrowth, so that affected tomato plants are larger than normal

and under commercial conditions of cultivation present an astonishing appearance of leafiness and abnormal extra growth. The fruit may be normal in appearance, though poor in quality,



FIG. 38. Tomato plant affected with *Nicotiana Virus 1A* (distorting or emanation virus). Note the filiform leaves and the distortion produced.

but the writer has frequently observed cases in which the fruits are conical and strongly corrugated. There is also a tendency for the fruit to fall prematurely or to fail to set. This disease was recorded for the first time in the British Isles from Scotland in

1935, and since then it has been observed in various localities, notably Ipswich. A similar disease has also been described in Russia (72). The virus differs in its reaction from the type virus in producing local lesions without systemic infection in White Burley tobacco plants. The writer has, however, produced systemic infection of White Burley tobacco with another stock of what appeared to be the same virus strain. In this case the symptoms in the tobacco were of the same distorting character. It is possible, of course, that here a slightly different strain of the virus was concerned.

It will be noticed that it is a property both of the type virus and the distorting strain to produce leaf enations, but whereas the former produces these outgrowths only on *Nicotiana paniculata* and *N. tomentosa*, the latter produces them on tomato and probably other plants. This virus strain gives the local lesions on *Nicotiana glutinosa* which are also characteristic of the type virus (1A, 83).

NICOTIANA VIRUS 1B. Smith

Synonym. *Tomato Yellow Leaf Virus.*

The disease caused in the tomato by this strain was first recorded by the writer in 1935 from some glasshouses in Ipswich (88). In young affected plants there is a strongly marked mottle on the youngest leaves; these are of an unusually dark green with pale areas in the region of the veins. There may be a certain amount of leaf distortion. In older plants, the middle leaves are very dark green in colour with some light green mottling. The lower leaves show the pronounced yellowing which is so characteristic a feature of the disease. These yellow leaves frequently show a banding of the veins with a dark green colour which stands out strongly against the yellow background (see Fig. 39, C). Necrotic areas and patches may also be present. Exposure to strong sunlight produces markings on these leaves of a peculiar purple-bronze colour. As the plant grows the yellowing spreads upwards until the whole plant, with the exception of the topmost leaves, is yellow. At this stage the disease strongly resembles a chlorosis induced by a nutritional deficiency such as nitrogen starvation, and indeed has been several times diagnosed as such. The fruit gives an appearance of having failed to ripen, the normal red colour being mottled with patches of yellow or pale green.

On tobacco and *N. glutinosa* the virus gives local lesions only, with no systemic spread (see Fig. 39, A).

In experiments on the seed transmission of this virus, the



D



writer obtained two apparently authentic cases of such transmission out of a very large number of seedlings. It is possible that these two cases can be explained on the basis of a mechanical contamination of the cotyledons, during germination, by virus on the outer seed coat (see also p. 235).

NICOTIANA VIRUS 1C. Bewley

Synonyms. *Tobacco Virus 6* (McKinney), Johnson; *Yellow Tobacco Mosaic Virus*, McKinney, 1926; *Tomato Aucuba Mosaic Virus*, Bewley.

By cross immunity tests Kunkel has shown that this common virus is a strain of the type virus and is not a different entity. Although the two produce very similar symptoms on a number of plants, they can be distinguished by their reactions on two differential hosts, *Nicotiana sylvestris* and *N. tabacum*. On *N. sylvestris* the type virus produces only faint chlorotic spots on the inoculated leaves. This is followed by systemic invasion of the plant by the virus, "clearing" of the veins of the youngest leaves and mottling of the other leaves. The aucuba mosaic virus produces necrotic lesions on the inoculated leaf without any further spread of the virus (54). Since the aucuba mosaic virus is of some importance in England to tomato growers, an account is given of the disease caused in that plant. First signs of infection appear on the young developing leaves of the crown about the fifth day after inoculation. These show a downward curling of the whole leaf, with slight turning down of the margins, and the surface of the leaf is rough, wrinkled or corrugated. By the seventh or eighth day points of chlorosis appear on these curled leaves, and by the twelfth or thirteenth day, when usually six more leaves have unfolded, the signs of disease differ in the different leaves. In extreme cases almost the whole surface of later developing leaves is pale yellow to white, with here and there

FIG. 39.

- A. Local lesions on leaf of tobacco, White Burley, caused by *Nicotiana Virus 1B* (tomato yellow leaf virus).
- B. Leaf enations on tomato caused by *Nicotiana Virus 1A* (enation virus).
- C. Symptoms on mature tomato plant caused by *Nicotiana Virus 1B* (tomato yellow leaf virus).
- D. Leaf hair from tobacco plant infected with *Nicotiana Virus 1C* (tomato aucuba mosaic virus): note the intra-cellular inclusion in lower cell.
(B, after Ainsworth; D, after Sheffield.)

small islets of intense dark green, which stand up as small blisters. In less extreme cases the green areas are larger, but as a rule the



FIG. 40. Leaves of tomato plant affected with *Nicotiana Virus 1C* (tomato aucuba mosaic virus).
(After Henderson Smith.)

area of white or pale yellow is greater than the green area. When the plant reaches a height of 18 to 24 inches, by which time the first flowers are forming, a typical leaf will show on each leaflet

areas of four different shades. Most of the surface is green, partly of normal tint and partly of a deeper, richer shade. Scattered over the leaf are patches of white and patches of yellow, irregular in shape and size, often angular and occurring in all parts of the leaf (Fig. 40).

The plant is not killed, but growth is checked ; compared with normal plants of the same age it is stunted and of spindling habit. There is no necrosis. The fruit may be normal in appearance or it may be mottled (78) (see Fig. 47, C).

Histopathology. The formation of intracellular inclusions, or X-bodies, is a common feature of diseases caused by *Nicotiana Virus 1* and its strains, and an account is given here of the X-bodies found in tomato plants affected by *Nicotiana Virus 1C* (tomato aucuba mosaic virus) (see Fig. 39, D). At about the time of appearance of macroscopic symptoms the cytoplasm becomes more conspicuous, its streaming is accelerated and innumerable minute particles appear in it ; by successive fusions these increase in size. They are carried about the cell by the plasm. By aggregation and fusion large protein masses are built up. As they become larger, their speed of movement about the cell decreases. In the course of a few days, practically all the particles are contained within a single mass, which finally becomes more or less rounded. In the hair cells it usually retains a granular appearance, but in other tissues it may become more homogeneous. As in other hosts, a spike-like crystal appears. Very occasionally a third type of inclusion body is found ; this consists of large hexagonal crystalline plates or of irregular polygonal plates which appear to be built up from a number of hexagonal ones of varying sizes lying edge to edge. These inclusions appear to be identical with the striated bodies commonly associated with tobacco mosaic diseases. Inclusions are formed quite abundantly in the cells of affected tomato plants. Their diameter may reach about 25μ in the basal cells of the hairs, but usually they are considerably smaller. They are formed in most of the hair cells of an infected leaf irrespective of green and chlorotic areas. When, as occasionally happens, the leaves of the host assume the "fern leaf" condition, intracellular inclusions are particularly well developed in the tegumentary tissues (76).

Sheffield (77) has studied the histology of the necrotic lesions produced by *Nicotiana Virus 1C* on the leaves of *N. glutinosa*. She finds that about twelve hours after inoculation a band of necrotic material begins to form within the cell wall, usually

between the lower epidermis and the spongy parenchyma. This band extends both laterally and towards the upper side of the leaf. At the same time, nuclear division is observed in the spongy parenchyma cells, but karyokinesis is not followed by cell division. As the necrotic meshwork extends the cells within it die and dry out. After about three days the lesion consists of a meshwork of this necrotic material. The cells are all dead and the virus is isolated within the necrotic area, all interchange between the infected and healthy parts of the leaf having been prevented. Certain other species of *Nicotiana* while reacting with local lesions, develop subsequently a systemic necrosis of veins, petioles and stem, which may cause the death of the plant. Such are *N. alata* Link. and Otto, *N. Sanderæ* Sander, *N. acuminata* and *N. langsdorffii*.

NICOTIANA VIRUS 1D. Holmes

Holmes has described a strain of tobacco mosaic virus which produces no symptoms when tested on a range of host plants, although it multiplies within them. This masked strain was isolated under artificial conditions at a high temperature from a distorting strain of the type virus (40). It produced no symptoms on six species of *Nicotiana*; these were *N. tabacum*, four varieties, *N. palmeri* A. Gray, *N. longiflora* Cav., *N. suaveolens* Zehm., *N. sylvestris* Spegaz. and Comes, and *N. trigonophylla* Dun. *Solanum nigrum* L., var. *nodiflorum*, which shows an obscure mottling with other strains of this virus, gave no perceptible reaction with the masked strain. *Nicotiana glauca*, which is capable of acting as a symptomless carrier of the type virus, showed no symptoms when inoculated with the masked strain, although virus increased at the site of inoculation and spread to some extent to non-inoculated leaves. On *N. glutinosa* the masked virus strain produces visible local lesions very similar in appearance to those produced by other non-masked strains. Since infection of a plant with one strain precludes entrance of another strain of the same virus, it is possible that this masked strain of virus may be used to protect tobacco and tomato plants from infection by other and more injurious strains. Holmes has indeed made some preliminary tests of this by comparing the yield of tomato plants infected with the masked strain with that of non-inoculated tomato plants, both lots being grown under conditions of exposure to natural infection with other strains of the virus. The results

of this preliminary trial seem sufficiently encouraging to warrant further investigation of such protective "vaccination."

The symptoms of twelve strains of *Nicotiana Virus 1*, representative of the range of symptoms produced by fifty-five strains, on tobacco, *Nicotiana sylvestris* and *N. glutinosa*, have been recently described (43).

One strain, derived from a slow-moving, necrotic-type strain on tobacco, killed tomato plants. Two strains produced unusually small lesions on leaves of *N. glutinosa*. Single pin-puncture inoculations of some strains to young tobacco plants produced as high as 50 per cent infection, whereas other strains were transmitted in less than 1 per cent of the attempts. Other strains ranged between these two extremes. All strains tested were found to withstand ten-minute exposures to a temperature of 80° C.

It will be of interest here to consider the work of Stanley in relation to strains of *Nicotiana Virus 1* and to compare the protein content of plants affected with the various strains with that of plants affected with the type virus. Stanley (92) has isolated two high molecular weight crystalline proteins from Turkish tobacco plants infected with *Strain 1C* (aucuba) and *Strain 1D* (masked strain). These two proteins, although apparently related to each other and to the crystalline protein from plants infected with the type virus, have been found to possess physical and chemical properties that distinguish them from each other. Altogether four different strains of *Nicotiana Virus 1* have been found to give rise to four different crystallizable, high molecular weight proteins. These results indicate that when *Nicotiana Virus 1* mutates and gives rise to a new strain, this change is accompanied by the production of a new protein (93).

NICOTIANA VIRUS 2. Johnson

Synonym. *Speckled Tobacco Mosaic Virus*, Johnson, 1927.

The Virus

Resistance to Chemicals. Withstands 50 per cent alcohol for one day, but is inactivated by 1 : 200 nitric acid in one hour.

Thermal Death-point. The virus is destroyed by ten minutes' exposure to 90° C.

Resistance to Ageing. The longevity *in vitro* of this virus is three or more months.

Transmission. The virus is sap-transmissible, but there is no information on the insect vector.

Host Range. There is little information on the diseases caused by this virus. On tobacco, the symptoms consist of mottling and speckling; on petunia, mottling, stunting, malformation and necrosis. On *Hyoscyamus*, henbane, the virus produces chlorosis, stunting and malformation; no symptoms are produced on *Nicotiana glutinosa*, pepper or pokeweed (47).

Geographical Distribution. The virus has only been recorded by Johnson from Wisconsin, U.S.A.

· NICOTIANA VIRUS 3. Johnson

Synonym. *Mild Tobacco Mosaic Virus*, Johnson, 1927.

The Virus

Resistance to Chemicals. Inactivation occurs after exposure to 50 per cent alcohol for one hour or to 1 : 200 nitric acid for a similar period.

Thermal Death-point. The virus is destroyed by exposure to a temperature of 60° C. for ten minutes.

Resistance to Ageing. Infectivity is lost after about six days' ageing *in vitro*.

Transmission. The virus is sap-transmissible, but the insect vector is not known.

Host Range. On tobacco this virus produces a very mild mottling, on *N. glutinosa* it gives rise to a mild chlorosis and stunting, on *N. rustica*, a mild mottling and malformation, while no symptoms are produced on *N. glauca* or pokeweed.

Geographical Distribution. The only record of this virus is that of Johnson from Wisconsin, U.S.A. (47).

NICOTIANA VIRUS 4. Johnson

Synonyms. *Tobacco Virus 8*, Johnson; *Tobacco Bleaching Mosaic Virus*, Johnson, 1927.

The Virus and its Transmission. The only information on this virus is contained in some notes by James Johnson (47). Its resistance to ageing *in vitro* is about three days and its thermal death-point is 75° C. at ten-minute exposures. It withstands 50 per cent alcohol, but is destroyed by 1 to 200 nitric acid in one day.

The virus is sap-transmissible, but there is no information on the insect vectors or on seed transmission.

Diseases caused by Nicotiana Virus 4

Solanaceæ

Nicotiana tabacum. On tobacco the virus produces a mosaic mottling with occasional chlorosis on young plants.

N. glutinosa. Unlike *Nicotiana Virus 1*, this virus becomes systemic in *N. glutinosa* with mottling and some malformation.

Physalis pubescens. On this plant the symptoms consist of chlorosis and mottling with some necrotic spots.

Phytolaccaceæ

On pokeweed, chlorosis, necrosis and mottling are produced.

NICOTIANA VIRUS 5. Böning

Synonyms. *Tobacco Virus 11*, Böning, Johnson's classification ; *Tobacco Stripe and Curl Disease Virus*, Böning, 1928.

The Virus and its Transmission. The virus is sap-transmissible, but the insect vector does not appear to be known. There is no evidence of transmission by the seed, but the virus is said to be carried in the soil, into which it is presumably introduced with compost from diseased stems. There is no information on the properties of the virus.

Disease caused by Nicotiana Virus 5

Solanaceæ

Nicotiana tabacum. Tobacco. The first symptom induced by artificial inoculation is a mottling of the upper portion of the plant, followed by necrosis of the young leaves and of the stem.

On the stems and veins the injuries consist of elongated, not always continuous, brown to slate-grey stripes of diseased and sunken tissue. On the stems several stripes, 0·5 to 1 cm. in width, may run parallel to each other. On the main veins the stripes are narrower, but otherwise similar to those on the stems. The death and collapse of the veins while growth is still proceeding result in a check to their growth relative to that of the intervening tissue, so that the latter bulges and the leaves present a curly or crumpled appearance with very sinuate margins, and are partially or wholly deformed. The necroses are first indicated by a pale mosaic colour, which subsequently turns yellow and ultimately brown.

Histopathology. The pith of infected stems is discoloured and shrivelled and cavities are formed in the dead tissue. Microscopic examination shows that the most severe damage occurs in the cells of the pith and cortical parenchyma, which are traversed in a tangential direction by necrotic zones. The decay spreads to the secondary cortex and xylem.

Host Range. The virus has been transmitted experimentally to *Nicotiana macrophylla*, *N. rustica* and several varieties of tobacco. It is apparently not transmissible to potato or tomato.

Geographical Distribution. The only record of this virus appears to be from Bavaria (17).

NICOTIANA VIRUS 6. McKinney

Synonyms. *Tobacco Virus 12*, McKinney, J. Johnson's classification; *Tobacco Green Mosaic Virus*, McKinney, 1929.

The Virus and its Transmission. McKinney (59) has isolated a virus causing a dark green mosaic in tobacco in the Canary Islands. The virus is sap-transmissible and resists desiccation, but there is no further information on its properties and it appears to be differentiated largely on a symptom basis.

Diseases caused by Nicotiana Virus 6

Nicotiana tabacum. Tobacco. The first indication of disease consists of a bleaching of the veins. The leaves may become slightly stunted and more or less deformed. Sometimes a very faint green mottling may be noted at this period, but in most cases no mottling is evident. The later leaves show less deformity. They are normal green in colour and no mottling can be detected. From three to five such leaves may develop. These are followed by three to five leaves which show very small, faint, light green mottlings. This condition becomes more intense on subsequent leaves until a very pronounced mottling occurs. This mottling is characterised by very large light and dark green areas and sometimes more than half of the leaf is solid dark green and the remainder solid light green. The margins of the leaves frequently turn downwards, giving an inverted spoon effect. This stage may be followed by one in which a few leaves develop very small, faint, light and dark green mottlings, as in the case of the earlier stage. This virus does not immunise tobacco plants against infection with yellow strains of *Nicotiana Virus 1* (see p. 280). It was found occurring naturally on *Nicotiana glauca*, on which it

produces similar symptoms. The virus does not appear to be transmissible to tomato.

NICOTIANA VIRUS 7. E. M. Johnson

Synonyms. *Tobacco Virus 13*, Johnson, E. M., J. Johnson's classification; *Tobacco Etch Virus*, Johnson, E. M., 1930.

The Virus and its Transmission. There is little information available on the properties of this virus. It is sap-transmissible, but except for grafting no other mode of transmission is known. The virus is unable to withstand desiccation. The host range seems largely confined to the Solanaceæ.

Diseases caused by Nicotiana Virus 7

Nicotiana tabacum L. Tobacco, var. Burley. The leaves of the growing point of affected plants have small chlorotic spots, $\frac{1}{16}$ to $\frac{1}{4}$ inch in diameter, in the interveinal areas. The margins of these spots become either partly or entirely necrotic. The lower leaves of plants infected in the field develop necrotic veins, which have an ochre-yellow to rusty-brown colour. The first sign of infection usually appears about six days after inoculation on rubbed leaves, and on the younger unrubbed ones, in the form of fine necrotic etching on the upper surfaces. A few days later, chlorotic spots appear in the bud leaves and these later become necrotic. Symptoms may continue to develop in this sequence for some time and then leaves that show only occasional chlorotic spots may develop for several weeks. If such plants are kept in a vigorously growing condition for a sufficient time numerous chlorotic and necrotic spots may again appear (46).

Lycopersicum esculentum. Tomato. Very slight symptoms are caused in tomato by this virus. There is a slight hooking back of the tips of the leaflets, together with an inrolling of their margins. The tissue between the veins is of a paler green colour and the lower leaves have a chlorotic cast not present in healthy plants. The leaves are smaller than normal.

NICOTIANA VIRUS 8. Johnson

Synonyms. *Tobacco Virus 18*, J. Johnson's classification; *Tobacco Streak Virus*, Johnson, 1936; *Tobacco Vein-streak Virus* (?), Jochems, 1938.

The Virus

Resistance to Ageing. The longevity *in vitro* is short, the virus is inactivated after twenty-four to thirty-six hours' ageing in extracted sap at a temperature of 22° C.

Thermal Death-point. The thermal death-point is 53° C. after exposure for ten minutes.

Dilution End-point. The virus barely survives a dilution of 1 in 20 and will probably not stand a dilution of 1 : 30.

Transmission. The virus is sap-transmissible and is moderately infectious, a high percentage of successful transmissions can be obtained by rubbing and by using young, recently infected plants as the source of inoculum.

The insect vectors are not known, but the virus is not transmitted by *Myzus persicae* Sulz., *M. pseudosolani* or *Macrosiphum gei*. There is no evidence of seed transmission.

Differential Hosts.

Local and systemic necrotic lesions are produced on tobacco, the latter being closely associated with the veins. Mottling and sometimes necrosis develop on *Nicotiana glutinosa* L.

Diseases caused by Nicotiana Virus 8

No evidence is available as to whether this virus is transmissible to plants outside the family Solanaceæ.

Solanaceæ

Nicotiana tabacum L. Tobacco. Under field conditions the disease is usually confined to the leaves, but not all the leaves on an affected plant show symptoms. The type of necrotic lesions produced is by no means uniform in character; spots, lines and circles with or without uniform patterns occur, more often on the basal half of the leaf than on the apical half. Vein-clearing, followed by "water-soaked" necrotic systemic symptoms, may appear on new leaves three days after inoculation, often preceded by local necrosis at the point of inoculation. Usually no mottling occurs either before or after the necrotic symptoms on tobacco. On young greenhouse plants, as in the field, the tendency for new leaves to recover is very striking, and this recovery does not appear to bear any relation to the external environment.

The virus is also transmissible to the following species of *Nicotiana*: *N. glutinosa*, *N. rustica* L., *N. tabacum* × *N. glutinosa* hybrid. Other susceptible Solanaceous plants are *Datura*

Stramonium L., *Nicandra physaloides* (L.) Pers and *Physalis pubescens* L. The following plants are immune from infection: tomato, *Lycopersicon esculentum* Mill.; potato, *Solanum tuberosum* L.; egg plant, *S. melongena* L.; and pepper, *Capsicum annuum* L.

Geographical Distribution. The disease has been recorded from Wisconsin, U.S.A. (48), and what is probably the same disorder has been recorded by Jochems (45) from Sumatra under the name of "tobacco vein-streak."

NICOTIANA VIRUS 9. Jochems

Synonyms. *Tobacco Virus 17*, Jochems, Johnson's classification; *Tobacco Rotterdam B-disease Virus*, Jochems, 1983.

The Virus and its Transmission. The virus is sap-transmissible and appears to be highly infectious. There is no information available on insect transmission, but the virus may be conveyed from one site to another by the soil. It passes a Berkefeld filter only with difficulty, and this is a point of difference from the tobacco mosaic type viruses, which easily pass such a filter. The host range seems to be restricted.

Diseases caused by Nicotiana Virus 9

Solanaceæ

Nicotiana tabacum. Tobacco. The earliest symptoms appear on the youngest leaves as scattered, indefinite light green or yellow areas in which irregular, dead brown spots, up to 5 mm. in diameter and either sharply limited by or elongated along the veins, develop after one or two days. The leaves become blistered, show irregular margins, turn yellow, and hang down. Besides this leaf mottling and distortion the pith and vascular ring of the stem show large, brown necrotic areas. Similar lesions subsequently develop also in the cortex, over which the epidermal cells collapse, causing depressions in the stem. The whole stem turns black, the leaf veins wrinkle and turgescence is lost some days before the death of the plant. A characteristic feature of advanced stages of the disease is the so-called "chambering" of the pith (division of the cavity by discs of tissue). When almost full-grown plants are infected, the veins and mid-rib turn black and wrinkle; eventually the leaves fade and drop. New leaves developing in these large plants during the eight- to ten-day incubation period show small necrotic or chlorotic areas over their entire surface

and do not attain a size of more than 10 cm., while the newly developed stem portion shows neither internal nor external necrosis, and the plant flowers and produces normal seed, but only reaches half the usual height. The only other host known for this virus is *Nicotiana sylvestris* (44).

Geographical Distribution. The disease has been recorded from Sumatra, where it has been prevalent since 1926 on the alluvial sandy loam or clay soils of the Deli coastal plain.

NICOTIANA VIRUS 10. Storey

Synonyms. *Tobacco Virus 16*, Storey, Johnson's classification ; *Tobacco Leaf-curl Virus*, Storey, 1932 ; *Tobacco Cabbaging or Crinkle Virus*, Storey, 1932 ; *Tobacco Kroepoek Virus*, Thung, 1932 ; *Tobacco Crinkly Dwarf Virus*, Moore, 1933 ; *Tobacco "Gila" Virus*, Jochems, 1926.

The Virus and its Transmission. The virus is not sap-transmissible and in consequence nothing is known of its properties. It is not seed-borne. The insect vector is the white-fly (*Aleyrodidae*) *Bemisia gossypiperda* (see p. 499). The host range seems to be fairly wide.

Diseases caused by Nicotiana Virus 10

Solanaceæ

Nicotiana tabacum. Tobacco. *Tobacco Leaf-curl* (see Fig. 41). The most characteristic symptom of tobacco leaf-curl disease is the production of leafy outgrowths from the veins on the lower surface of the leaves (cf. *Nicotiana Virus 1*, p. 241) ; these are sometimes up to a centimetre in width, but usually amount to no more than a dark green thickening of sections of the veins. Combined with this feature is a stunting of the whole plant and twisting and curling of the leaves. The manifestation of the disease varies greatly

FIG. 41. *Nicotiana Virus 10* (tobacco leaf-curl).

- A. Portion of the underside of a leaf of infected tobacco, var. Amorello, showing enations and irregular thickening of the finer veins.
- B. Portion of tobacco leaf magnified to show clearing or yellowing of the finer veins.
- C and D. Tobacco plants affected with the disease in the field. (The symptoms shown in A and B are important diagnostic characters.)

(A, after McClean ; B-D, after Pal.)



according to environmental conditions and the variety of tobacco concerned. Chlorosis or necrosis is not characteristic of the disease (95). Thung (102) differentiates three types of leaf-curl (*kroepoek*) : (1) The *common kroepoek*, in which the leaf edges are curled in places towards the dorsal side, and show thickening and outgrowths (enations) of the veins ; (2) *curl disease* (*krulziekte*), characterised by the curling of the whole leaf edge towards the dorsal side, with enations of the veins, the lamina arching towards the ventral side between the finer veins, the distance between the latter being much reduced ; and (3) *transparent kroepoek*, distinguished by the curling of the leaves towards the ventral side, and the clearing of the veins, enations being absent.

Histopathology. Histological examination of the diseased tissues shows that in the curl diseases there are more layers of densely aggregated spongy parenchyma cells with fewer inter-cellular spaces than normal. "Common" *kroepoek* is characterised by a disturbance of sugar transport throughout the leaf ; an increase of the primary phloem in the veins ; the enlargement of the pericycle through cell division ; the formation of new woody vessels surrounded by a cambium, so that new steles arise inside the old pericycle ; and the loss of dorsiventrality in the leaf through the substitution of palisade for spongy parenchyma. Furthermore, palisade parenchyma and stomata are formed in the lobed veins of the affected tobacco leaf ; the lobes afterwards develop into secondary leaflets (enations) in which the new steles proceed in reverse orientation. These new leaflets lie with the morphological underside against the underside of the old leaf.

The "transparent" type of disease produces in the leaf-veins typical swelling of the ends of the xylem vessels and sieve-tubes and enlargement of the pericycle and cortical parenchyma cells. The sieve-tubes are curved and the cell walls of the primary phloem as well as those of the pericycle are irregularly swollen. There is much secondary phloem still apparently functioning. Some doubt exists as to whether "transparent" *kroepoek* may not be due to a separate virus, and Kerling (52) suggests the possibility of two viruses being concerned. In conclusion, the primary diagnostic features of the disease in tobacco seem to be the enations and the simple greening of the veins rather than the curling of the leaves, which may arise from a variety of other causes (96).

Other species of *Nicotiana* susceptible to infection with this virus are *N. glauca* and *N. rustica*.

Lycopersicum esculentum. Tomato. The tomato plant has

been infected artificially by grafting a scion from a leaf-curl tobacco plant. The symptoms produced in the tomato are essentially similar. The wild tomato has been observed in Nyasaland apparently naturally infected with this virus.

Compositæ

Zinnia elegans Jacq. The most characteristic symptom in the zinnia is the thickening of the lower surface of the veinlets, preceded by curling of the leaf blades. Infection begins at the growing points of the younger leaves and disturbs the normal course of development, so that diseased plants often fail to attain a height of more than 1 foot and the flowers are dwarfed, of a poor colour, and partially sterile. During the warm damp season the axillary buds are forced into growth and form stunted shoots with small crinkled leaves, often massed together in a rosette or bunched form (63). Infected zinnia plants show loss of dorsiventrality in the leaf through the formation of a loose, irregular palisade parenchyma with large intercellular spaces. Other members of the Compositæ susceptible to the leaf-curl virus are the weeds *Vernonia* spp. and *Synedrella nodiflora*. The hollyhock *Althaea* (Malvaceæ) also appears to be susceptible. Symptoms suggestive of leaf-curl have been observed in Nyasaland upon *Crotalaria usaramoensis* and Biloxi soya beans (Leguminosæ).

Geographical Distribution. The distribution of the leaf-curl virus is wide in tropical countries. It has been recorded from India (Dehra Dun), Nyasaland, Rhodesia, South Africa, Tanganyika, Java, Madagascar, Zanzibar and the Belgian Congo. The disease has also been recorded from Roumania.

Control. There appears at the moment to be little chance of controlling tobacco leaf-curl by the attempted elimination of the insect vector, and control measures are better applied to the eradication of wild host plants of the virus, especially *Vernonia* spp., and the removal of the old stumps of infected tobacco plants.

NICOTIANA VIRUS 11

Synonym. *Tobacco Necrosis Virus*, Smith and Bald, 1935.

The Virus

Resistance to Chemicals. This is the most resistant plant virus so far described. It is unaffected by alcohol and is still infectious after six months' storage in absolute alcohol at laboratory temperature.

Effect of Enzymes. The virus is unaffected by the proteolytic enzymes pepsin, trypsin and papain.

Serological Reactions. The virus gives no antigenic reactions under conditions in which *Nicotiana Virus 1* (tobacco mosaic virus) and *Solanum Virus 1* (potato virus X) are strongly antigenic.

Thermal Death-point. The thermal death-point in extracted sap is 72° C., but the virus is not entirely inactivated after fifteen minutes' exposure to 100° C. dry heat.

Dilution End-point. The virus has a dilution end-point of 1 : 10,000.

Longevity in Extracted Sap. The resistance to ageing in extracted non-sterile sap is about twenty days.

Resistance to Desiccation. The virus can be completely dried over sulphuric acid, without losing infective power. It retains its infectivity in the dried state for three months or longer.

Particle Size. The average particle diameter as measured by Elford's gradocol membranes is 20 to 30 millimicrons.

Effect of Ultra-violet Light. The virus appears to be inactivated after twenty minutes' exposure, in a gelatine film, to the open arc.

Effect of γ -rays. The virus is unaffected by exposure to γ -rays for five days at a temperature of 23° C. and 2,000 r units per hour.

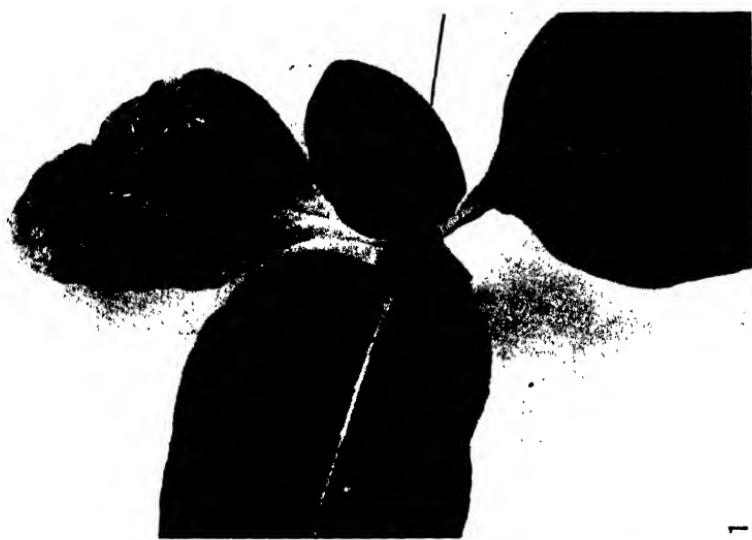
Transmission. This virus spreads in a manner which is unique among plant viruses; it is water-borne and also air-borne to the soil, whence it makes its way to the roots of the plant. It is not seed-borne, nor do insects play any part in its transmission. The virus is sap-transmissible, but owing to the fact that with one exception it is not systemic in its host, it cannot be transferred by grafting (84, 85, 86, 87).

Differential Hosts

The French or snap bean (*Phaseolus vulgaris*) is a very sensitive indicator plant for the virus. Reddish local lesions develop on the inoculated leaves within forty-eight to seventy-two hours. These

FIG. 42.

- A. Natural infection of *Nicotiana Virus 11* (tobacco necrosis), on tobacco, White Burley: note necrosis is confined to lower leaf.
- B. Local lesions on bean (*Phaseolus vulgaris*) var. Canadian Wonder, produced by artificial inoculation with *Nicotiana Virus 11*.
- C. Systemic infection of snapdragon (*Antirrhinum* sp.) with *Nicotiana Virus 12* (tobacco ringspot virus).
(C, after Wingard.)



A



B



C

lesions gradually spread, picking out the vascular system as they progress (see Fig. 42).

Diseases caused by Nicotiana Virus 11

The virus is very unusual in its behaviour in that normally it is confined to the roots of a large number of plants without producing any visible signs of its presence. The tobacco plant and *N. glutinosa* are the only plants in which naturally induced symptoms of disease have been observed. The virus appears to be confined to glasshouses.

Solanaceæ

Nicotiana tabacum. Tobacco, vars. White Burley, Virginia, Turkish, Maryland, Ambalema, etc. The first sign of natural infection in the tobacco plant is the appearance of necrosis running along the mid-ribs and smaller leaf-veins of the lowest leaf or pair of leaves (see Fig. 42, A). In very young seedlings this necrosis rapidly becomes complete and the plants are killed. In older plants the necrosis is usually confined to the lowest, and consequently the oldest, leaves. These leaves become entirely necrotic, dry up and the plant is then normal in appearance and remains so throughout its life. The virus is, however, invariably present in the roots of such plants as well as in the roots of a high percentage of normal-looking glasshouse-grown tobacco plants which never show disease symptoms. The virus produces only necrotic symptoms, mottling or chlorosis due to this virus has never been observed on any host plant.

When the virus is applied mechanically to the leaves of tobacco by rubbing, the initial symptoms differ from those of a natural infection. Numerous separate circular or ring-like necrotic spots develop on the inoculated leaves; these spread until the whole leaf is necrotic, but there is seldom any movement of the virus beyond the inoculated leaf.

N. glutinosa. The reactions of this plant are essentially similar to those of tobacco, though *N. glutinosa* seems less sensitive to artificial inoculation and the lesions so induced are smaller and not so numerous.

Other Host Plants of the Virus. *Nicotiana Virus 11* has been found occurring naturally in the roots of a number of different types of plants growing in the glasshouses at Cambridge. These plants, however, show no visible signs of infection. The virus has been isolated from the roots of tomato, aster, zonal pelargonium, *Primula obconica*, *Polyanthus*, and *Phaseolus vulgaris*.

The range of host plants in which the virus will produce local lesions when applied experimentally is very wide. Such symptoms have been produced in the following plants :

Cruciferæ. *Raphanus* sp.
Polygonaceæ. *Rumex obtusifolius*. *Polygonum convolvulus*.
Chenopodiaceæ. *Spinacia oleracea*. *Chenopodium album*.
Cucurbitaceæ. Seedling cucumber plants.
Grossulariaceæ. *Ribes* sp.
Compositæ. *Carduus* sp. *Sonchus oleraceus*.
Leguminosæ. *Phaseolus vulgaris*, *Vigna sinensis*.
Solanaceæ. *Nicotiana tabacum*, *N. glutinosa*, *Lycopersicum esculentum*, *Datura Stramonium*.

Systemic infection with this virus is produced only in the French bean (*Phaseolus vulgaris*), and that was not observed until the virus had been associated with this plant in inoculation experiments for eighteen months. There is, therefore, some evidence that the reaction of the French bean to the virus has undergone slight modification. Inoculation to the leaves produces numerous small red necrotic lesions which develop about forty-eight hours after inoculation. The virus then spreads through the leaf, etching out the vascular system as it goes (Fig. 42, B).

In the other plant species there is very little spread from the inoculated leaf. In *Polygonum* occasional isolated foci of infection develop on leaves some distance from the inoculated leaf, as if a few virus particles travel along and multiply at various points in the length of this climbing plant.

Geographical Distribution. In addition to the Cambridge record of this virus, the writer has been informed of its occasional occurrence in glasshouses at Harpenden and Cheshunt, Herts, in Edinburgh and in Adelaide, South Australia. It has never been isolated from the roots of susceptible plants growing out of doors, unless these plants had been raised as seedlings in the glasshouse.

Control. From the economic point of view *Nicotiana Virus 11* is of little importance, though its scientific interest is great. It can be eradicated by careful sterilisation of the soil and by the regular cleansing of the water tanks.

NICOTIANA VIRUS 12. Fromme

Synonyms. *Tobacco Virus 10*, Fromme, 1927, J. Johnson's classification; *Tobacco Ringspot Virus*, Fromme and Wingard; *Ringspot No. 1*, Price, 1936.

The Virus

Crystallization. Stanley and Wyckoff (94) claim to have isolated by means of an ultracentrifuge a high molecular weight crystalline protein from Turkish tobacco plants infected with *Nicotiana Virus 12* possessing the properties of *Nicotiana Virus 12*, and differing markedly from the protein of *Nicotiana Virus 1* in its physical, chemical and serological properties.

Reaction with Chemicals. Safranin appears unable to precipitate this virus and may possibly destroy it. It can be precipitated with acetone, but with considerable loss in virulence; 95 per cent ethyl alcohol can be used similarly, but the precipitate does not form so readily. It retains infectivity for twenty-four hours in 60 per cent ethyl alcohol, but is inactivated after twenty-four hours in 70 per cent alcohol.

Photo-dynamic Action of Methylene Blue. The virus is completely inactivated by the photo-dynamic action of methylene blue. Ten cubic centimetres of the virus-dye mixture, at pH 5.8 to 6, when exposed for two minutes to a 500-watt lamp, gave a negative reaction when inoculated to tobacco (16).

Serological Reactions. *Nicotiana Virus 12* does not produce very potent antiserum by inoculation to rabbits and the complement fixation test is negative. The precipitin reaction, however, shows this ringspot virus to be a separate and distinct entity (18).

Thermal Death-point. Inactivation ensues after five to ten minutes' exposure to 60° C. and after three minutes at 70° C. (33), but not after ten minutes at 55° C.

Dilution End-point. Very slight infection is obtainable at dilutions of 1 : 1,000, and at 1 : 10,000 only an occasional trace.

Longevity in Extracted Sap. The virus retains infectivity in extracted sap after ageing for three days at room temperatures, but is inactivated after four days. It retains infectivity, however, for twenty-two months at a temperature of -18° C.

Desiccation. The virus appears unable to withstand drying and completely dry infected leaves give no reaction when inoculated to susceptible plants.

Filtration. The virus passes a Berkefeld filter of V grade easily, it is largely held back by one of N grade and only a trace of infection is obtained after passage of a W grade filter. The writer has filtered the virus through Elford's gradocol membranes and has found that it passes a membrane of 0.15 μ average pore diameter, but is held back by membranes below that grade. Thornberry, on the other hand, finds that the virus passes a

membrane of $0\cdot045\mu$, but is retained by one of $0\cdot038\mu$. He assigns to this virus a particle size of $0\cdot015\mu$ (15 millimicrons) (101).

Methods of Transmission. This ringspot virus is sap-transmissible to the majority of its susceptible host plants. The insect vector is not known. The virus is not carried in the seed of tobacco (*cf. Nicotiana Virus 13*), but is transmitted through the seed of *Petunia* sp. Out of 810 petunia seedlings tested, 160, or 19·8 per cent, developed the ringspot disease (31, 33).

Differential Hosts

Nicotiana tabacum. Tobacco. Well-defined necrotic rings with a central spot are produced by this virus in the tobacco plant. There is at first considerable necrosis and stunting, followed by a rather rapid disappearance of symptoms.

Vigna sinensis. Cowpea. Primary lesions in the form of solid necrotic spots develop on the inoculated leaves. These are followed by systemic necrosis, and the plant is usually dead on the ninth day following inoculation (compare these symptoms with those caused by *Strain A and B* of this virus and by *Nicotiana Virus 13*) (71).

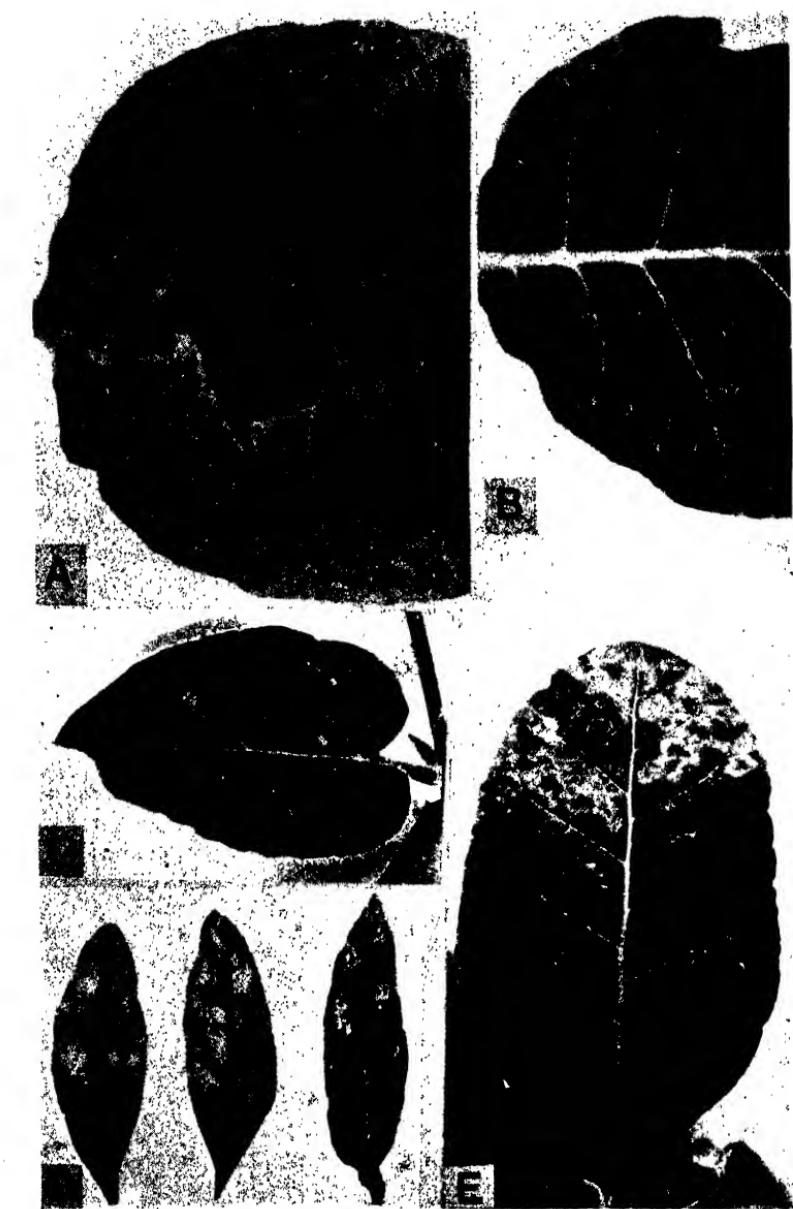
Diseases caused by Nicotiana Virus 12

Nicotiana Virus 12 has a very wide host range, and it is not practicable to describe every disease produced by it. It has been found infecting, or has been transmitted experimentally to, plants of thirty-eight genera, representing seventeen families. A selection has, therefore, been made of the more important diseases caused by this virus. A characteristic of this and other ringspot viruses is the ability of the affected plant to recover from the disease while still retaining the virus.

Chenopodiaceæ

Spinacia oleracea L. Spinach. The first symptoms are those of systemic infection, large chlorotic areas appearing on the young leaves. These areas occasionally become bright yellow and show a marked tendency to necrosis, the entire affected region usually dying out rapidly. The leaves are not as a rule malformed.

Affected plants show a tendency to recover, especially in warm weather, and may produce leaves which are almost free of symptoms. As a rule spinach plants are not killed by this disease, although the entire foliage may become yellow and the plant much stunted.



Beta vulgaris. Garden beet. The leaves of affected beet plants show faint zigzag lines and patterns. These lines are of a lighter colour than the normal tissue, but are never necrotic.

Cucurbitaceæ

Cucumis sativus, cucumber; *Cucurbita pepo*, vegetable marrow, pumpkins; *Cucumis melo cantalupensis*, cantaloupe. These plants are very susceptible to the virus, symptoms developing freely within three days of infection. The young spots on the leaves consist of a small yellowish-brown, pin-point-like centre surrounded by a bright yellow margin or halo. The pin-point centre in some cases becomes necrotic, but as a rule the necrosis does not spread throughout the spot. Although this type of spot is the most characteristic for this group of plants, there is a tendency for definite rings to appear, especially in the case of experimental infections, on the inoculated leaves. In some cases the tiny brown centres are surrounded by two or three necrotic rings.

Infection becomes systemic in about ten days and the halo type of spot appears in great numbers on all the new leaves. In the case of the dipper gourd the leaf is literally covered with light yellow spots no more than 1 mm. in diameter. These tiny spots have definite pin-point-like centres that are "water-soaked" in appearance. On squash and nest-egg gourds the spots are much larger, but are of the same type. On pumpkin the spots are restricted primarily to the leaf-veins. The halo type of spot also appears on cantaloupe, cucumber and "dish-cloth" gourd, but, in addition to this, definite rings and zigzags often develop. These lines and zigzags are much lighter in colour than the normal green leaf-tissue, but as a rule there is no necrosis.

The fruits of cucurbits affected with this virus also show

FIG. 43.

- A. Leaf of infected tobacco plant showing symptoms caused by *Nicotiana Virus 12* (tobacco ringspot virus).
- B. Leaf of tobacco, White Burley, showing symptoms caused by *Nicotiana Virus 14* (Bergerac ringspot virus).
- C. Systemic symptoms in *N. glutinosa* caused by *Nicotiana Virus 14*.
- D. Symptoms on leaves of Jerusalem Cherry caused by *Nicotiana Virus 12*.
- E. Leaf of tobacco, White Burley, showing symptoms caused by *Nicotiana Virus 18* (Price's tobacco ring-spot virus No. 2).

(A, after Henderson; D, after Wingard.)

symptoms, and these have been described on nest-egg gourd and "Golden Summer" crookneck squash. The fruits show symptoms very early and often drop when only 1 or 2 inches in length. The symptoms first appear as small circular "water-soaked" spots not more than 1 mm. in diameter. They become depressed in a few days and give the surface of the fruit a pitted appearance. These pits become encircled by a very narrow line of "water-soaked" tissue in four or five days. The spots penetrate to a depth of 2 to 3 mm. as a rule and may reach all the way to the seed cavity. They develop a deep green pigment in contrast to the white or yellow pigment of the normal tissue. In mature fruits the spots, which at first are depressed, gradually become elevated and finally appear as pimples on the surface of the mature fruit. The spot in its final stage is composed of a small elevated centre surrounded by one or more definite rings (109) (see Fig. 44, C).

Leguminosæ

Vigna sinensis. Black-seeded cowpea. Necrotic spots develop on the inoculated leaves about five days after inoculation; these are followed soon after by infection of the leaf-veins and petioles. The veins turn black and shrivel, and the lesions on the petioles become dark and sunken, the whole infection somewhat resembling that of anthracnose. Dark sunken lesions also appear on the main stem, and the plants die in nine to fourteen days (109).

Phaseolus vulgaris. French bean, kidney or snap bean. Necrotic spots usually develop on the inoculated leaf about three days after inoculation. The spot has a bright centre 1 mm. in diameter which is surrounded by a band of dark brown tissue. The margins of the spot consist of newly killed tissue which has a black "water-soaked" appearance. After infection has become systemic, spots develop on the new leaves that are composed of rings of necrotic tissue intervening between zones of apparently normal tissue. This plant is frequently, but not invariably, killed by the virus (see Fig. 45, A).

Pisum sativum L. Garden pea. Symptoms produced by this virus on pea seedlings begin with top-necrosis and an occasional ring pattern on leaflets not killed. No mottle pattern is produced as in the case of *Pisum Virus 2*. The upper leaflets frequently present a netted, bleached appearance for a few days, and then collapse completely. A conspicuous brownish discolouration usually develops rapidly on the entire stem. A large percentage of inoculated plants die within two weeks of inoculation. No

primary local lesions have been observed on the inoculated foliage (97).

Melilotus officinalis Lam. Sweet clover. Dwarfing of the plants with a yellowing of the leaves seems to indicate infection. Leaves from diseased plants held to the light usually exhibit light coloured areas in the tissue and, in some cases, faint rings, but plants with chlorotic abnormalities in the leaves are not always infected with the ringspot virus. Dwarfing of otherwise apparently healthy plants, however, seems to be a very definite sign of infection (33).

Melilotus alba Desr. White sweet clover. Chlorotic areas develop on the leaves, often following the mid-ribs of the leaflets and extending along the lateral veins towards the margin. Some leaflets may show chlorotic blotches and spots of various sizes. Very often the markings are of fine lines that follow along the veins or run in irregular circles. The younger leaves are severely dwarfed and deformed, with much puckering and twisting of the bud leaves (32).

Compositæ

Zinnia elegans Jacq. Garden zinnia. No definite ring symptoms develop on the leaves of zinnia, but infection becomes systemic. The new leaves that develop after inoculation show a marked stunting and in some cases may be slightly crinkled, but no other symptom has been observed.

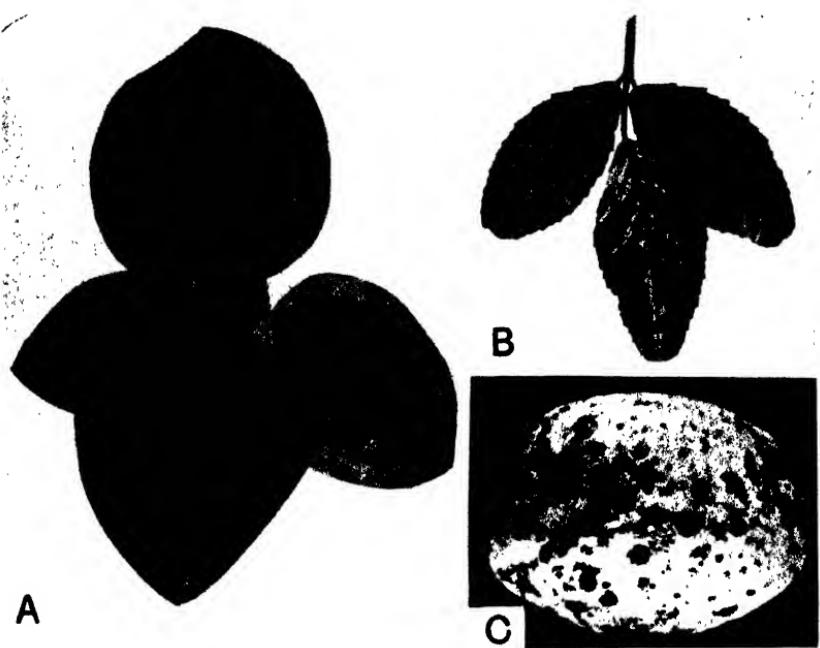
Callistephus chinensis Nees. China aster. Necrotic spots develop on the inoculated leaves about three days after inoculation. Systemic infection takes the form of rings or necrotic spots on the new leaves, as the plant continues to grow these symptoms gradually disappear. Affected aster plants are not killed by this virus.

Calendula officinalis L. Pot marigold. Symptoms take the form either of ring-like spots or fine zigzag lines on the leaves. The latter are the most frequent manifestation on this species. The lines are pale in colour and are not necrotic.

Bidens discoidea (T. and G.) Britton. Spanish needle. Symptoms on this species take the form of numerous very fine zigzag lines which occasionally form enclosures with irregular borders.

Violaceæ

Viola papilionacea Pursh. Common violet. **Viola tricolor* L. Giant pansy. Numerous necrotic rings with green centres develop on the leaves of infected plants of these two species.



Solanaceæ

Plants belonging to this family appear to be particularly susceptible to infection, especially many species of *Nicotiana*.

Nicotiana tabacum. Tobacco, vars. White Burley, Maryland, Turkish (see Fig. 43, A). Symptoms occur only on the leaves and are usually visible at the site of inoculation, within forty-eight to sixty hours. The first sign is the development of several single necrotic rings, which become blanched or brown in colour after a few days. A secondary ring or margin, 3 to 4 mm. in diameter, appears in about four days, and a third ring, 6 to 7 mm. in diameter, in about five days. This process continues when the spots are thinly distributed over the leaf, until finally there are several of these necrotic rings with alternating zones of normal-looking tissue. The centre of the spot consists of the primary ring and its island of encircled tissue. The tissue in the centre of the spot often dies in five or six days after the spot appears, especially if the primary ring is very small in diameter.

Infection becomes systemic in eight to fourteen days after inoculation. The symptoms of systemic infection show a marked tendency to follow the mid-rib and larger veins of the leaves, yet the characteristic circular spots appear in the interveinal tissue. About fourteen days after inoculation new leaves appear on which the symptoms may either fail to develop or will develop only slightly, appearing on the tip of the leaf alone. New leaves produced after this stage has been reached may appear normal or they may show a faint greyish mottling.

The leaves of plants on which the ringspot symptoms have become masked appear to be a little thicker and to have a more leathery texture than those of normal plants. This disappearance of symptoms is characteristic of ringspot viruses and is found in the diseases caused by *Nicotiana Viruses 13 and 14*, which are also of the ringspot type. Price (70) has shown that this recovery of tobacco plants from the ringspot disease is accompanied by a marked decrease in virus concentration and is followed by immunity from re-infection. Price considers that this constitutes

FIG. 44. *Nicotiana Virus 12* (tobacco ringspot virus).

- A. Systemic infection of Petunia.
- B. Symptoms on lucerne (alfalfa).
- C. Symptoms on mature fruit of *Nest Egg* gourd.
- D. Systemic necrosis produced in *Datura Stramonium*.
(A and B, after Henderson; C, after Wingard.)

an acquired immunity comparable in many respects to that which obtains in virus diseases of animals.

Intracellular Inclusions. Intracellular inclusions, or X-bodies, have been observed in both primary and secondary lesions of Turkish tobacco plants affected with this virus. A typical X-body is elongate, somewhat kidney-shaped, and tapering slightly toward the end opposite the cell nucleus. The main mass of the body is densely granular and contains several small vacuoles. The surface of the body also appears to be vacuolate. The vacuolation is such that on one side the body appears to consist of a central compact mass of granular material containing some small vacuoles, surrounded by a hyaline membrane-like portion. Some loosely aggregated granular material is irregularly distributed between the outer "membrane" and the inner compact granular mass. There are also present, rather uniformly distributed throughout the inclusion, numerous very small cuboidal bodies, apparently crystalline in character, which stain densely with safranin (110).

A number of other species of *Nicotiana* are susceptible to infection with *Nicotiana Virus 12*, and of these a certain number appear to be hypersensitive. These are *Nicotiana Clevelandii*, *glutinosa*, *longiflora*, *multivalvis*, *plumbaginifolia*, *quadrivalvis*, *repanda*, *Sanderæ*, *suaveolens*, *sylvestris* and *trigonophylla*. The most characteristic symptom of infection for this group is the development of spots which are necrotic throughout. The margin of the spot consists of newly killed tissue which has a black water-soaked appearance. This marginal tissue turns brown on drying and a new necrotic band is formed. Thus the spots enlarge until they finally coalesce and either kill the entire leaf or large sections of it.

Lycopersicum esculentum. Tomato. The virus is only with difficulty transmissible to the tomato by sap-inoculation. It can, however, be transmitted by grafting. About three weeks after completion of the graft small necrotic spots develop in the tomato leaves. A few of these may be in the form of rings, while others may be in the shape of short necrotic lines or dashes arranged end to end in a chain-like form. Sometimes large chlorotic rings are produced on the leaves (38).

Petunia. Infected petunia seedlings are severely dwarfed and stunted and the first few leaves are mottled in colour and streaked with watery green spots following the veins. Some leaves may show less of the watery green areas and more light

brown streaks which appear to be due to a slight necrosis of the tissues. Occasionally there is some curling of the leaves in places along the margin. For a time infected plants remain stunted, but later they seem to outgrow the symptoms to a certain extent; although isolated leaves may continue to show the chlorotic rings and markings typical of systemic infection. Flowering may be profuse, but the seed pods are markedly stunted and bear only a few seeds in comparison with normal pods. The virus is transmitted through the seed of this plant to the extent of about 20 per cent. (see Fig. 44, A).

Scrophulariaceæ

Antirrhinum majus. Snapdragon. The initial symptom of infection on snapdragon is the formation of numerous typical necrotic rings (see Fig. 42, C). The spots remain as alternating zones of living and necrotic tissue for about a week and then the entire spot becomes necrotic and the necrosis spreads until the spots coalesce and kill the entire leaf. The snapdragon is one of the species which is apparently killed outright by this ringspot virus.

Host Range. The following is a fairly comprehensive list of plants on which infection with *Nicotiana Virus 12* has been obtained or observed (109).

Solanaceæ

Nicotiana acuminata Grah.; *N. Clevelandii* A. Gray; *N. glutinosa* L.; *N. langsdorffii* Weinm.; *N. longiflora* Cav.; *N. paniculata* L.; *N. plumbaginifolia* Viv.; *N. quadrivalvis* Pursh; *N. quadrivalvis*, var. *multivalvis* Gray; *N. repanda* W.; *N. rustica* L.; *N. sanderae*; *N. suaveolens* Lehm.; *N. sylvestris* Speg.; *N. tomentosa* Ruiz. and Pav.; *N. trigonophylla* Dun.; and *N. tabacum* L. Various botanical and agronomic varieties of tobacco are also susceptible. *Datura Stramonium*, Jimson weed; *Nicandra physaloides* (L.) Pers., apple of Peru; *Petunia violacea* Lindl., petunia; *Physalis angulata* L., ground cherry; *Solanum carolinense* L., horse nettle; *Solanum melongena* L., var. *esculentum* Nees, egg plant; *Solanum nigrum* L., black nightshade; and *Solanum pseudocapsicum* L., Jerusalem cherry.

Cucurbitaceæ

Citrullus vulgaris Schrad., Monte Cristo watermelon; *Cucumis melo* L., var. *cantalupensis* Naud., Honey Ball and Honey Dew cantaloupe; *Cucumis sativus* L., Everbearing and Ideal White

Spine cucumber; *Cucurbita pepo* L., var. *condensa* Bailey, Golden Summer Crookneck and Mammoth White Bush squash; *Cucurbita moschata* Duschene, Cushaw pumpkin; *Cucurbita pepo* L., cornfield pumpkin; *Cucurbita pepo* L., var. *ovifera* Bailey, Nest Egg gourd; *Lagenaria leucantha* Rusby, Dipper gourd; and *Luffa cylindrica* Roem., Dish Cloth gourd.

Compositæ

Ambrosia artemisiifolia L., small ragweed; *A. trifida* L., giant ragweed; *Aster laevis* L., Ostrich Plume aster; *Bidens discoidea* (T. and G.) Britton, Spanish needle; *Calendula officinalis* L., pot marigold; *Callistephus chinensis* Nees, China aster; *Erigeron canadensis* L., field erigeron; *Helianthus annuus* L., mammoth Russian sunflower; *Lactuca sativa* L., var. *capitata* L., head lettuce; *L. scariola* L., prickly lettuce; *Tagetes erecta* L., African marigold; *Zinnia elegans* Jacq., garden zinnia.

Leguminosæ

Dolichos lablab L., broad Windsor bean; *Melilotus officinalis* Lam., sweet clover; *Phaseolus lunatus* L., small lime or Sieva bean; *P. vulgaris* L., French or kidney bean; *Medicago sativa*, *Trifolium repens*, and *Vigna sinensis* Endl., black-eye cowpea.

Chenopodiaceæ

Beta vulgaris L., garden beet and Silesian sugar beet; *B. vulgaris* L., var. *cicla* L., Swiss chard; *Chenopodium album* L., lamb's quarters, white goose-foot.

Violaceæ

Viola papilionacea Pursh, common violet; and *Viola tricolor* L., giant pansy.

Ficoidaceæ (Aizoaceæ)

Tetragonia expansa Murr., New Zealand spinach.

Amaranthaceæ

Amaranthus paniculata L., pigweed.

Convolvulaceæ

Ipomoea purpurea (L.) Roth., morning glory.

Cruciferæ

Barbarea Barbarea (L.) MacM., winter cress.

Euphorbiaceæ

Ricinus communis L., castor oil plant.

Labiatae

Salvia splendens Ker., scarlet sage.

Malvaceæ

Hibiscus esculentus L., okra.

Phytolaccaceæ

Phytolacca decandra L., pokeweed.

Polygonaceæ

Polygonum hydropiper L., smartweed.

Scrophulariaceæ

Antirrhinum majus L., snapdragon.

Geographical Distribution. This disease was first observed in tobacco crops in Virginia, U.S.A., in 1917. Since that time it has been recorded from practically all of the important tobacco-growing districts in the United States. What is possibly the same disease has been recorded from Australia, but there is apparently no record of the virus in Europe, though there are viruses which may cause similar-appearing diseases in tobacco.

NICOTIANA VIRUS 12A

Synonym. *Yellow Ringspot Virus*, Valleau, 1932.

The Virus and its Transmission. The disease of yellow ringspot on tobacco was first described by Valleau in 1932 (104), who suggested that this and the green ringspot (*Strain B*), dealt with in an ensuing paragraph, were both strains of the same ringspot virus (*Nicotiana Virus 12*). Further evidence on this point has been brought forward by Price (71), who shows that infection with *Strain A* protects a plant from further infection by both *Strain B* and the type virus. Studies on the properties of this virus have not been carried out, but there is no reason to suppose they differ in any material factor from those of the type virus.

Transmission. The virus is sap-transmissible, but unlike the type virus it appears to be carried in the seed of tobacco to the extent of 1 to 17 per cent.

Differential Hosts

Nicotiana Virus 12A may be recognised by the symptoms it produces on tobacco, which consist of bright yellow, as well as necrotic, systemic lesions. It is further characterised by the fact that old leaves of plants which have "recovered" from the disease (see p. 277) are yellow in colour, particularly along their margins.

Diseases caused by Nicotiana Virus 12A

Solanaceæ

Nicotiana tabacum. Tobacco. While the symptoms caused in tobacco by this strain of virus are of the same general order as those due to the type virus, they differ in their colour. Whole leaves of affected plants may bleach slightly or turn light yellowish-green to nearly white. On the young shoots or suckers a golden-yellow colour may develop with few or no necrotic rings. Seedlings from plants affected with this virus strain turn yellow soon after germination and have a bleached yellowish-green appearance throughout their growth. Seed transmission of this strain is, therefore, easily demonstrated (104). The early symptoms following artificial inoculation resemble those produced by *Strain B*, but are slower in developing. Necrotic single, double or triple rings appear on the older inoculated leaves and single rings or necrotic spots appear on the younger rubbed leaves. There is soon a marked tendency towards yellowing, especially in the new leaves. The yellowing may develop in small spots, which may increase in size, or chlorotic yellowish rings sometimes develop. The tissue in the vicinity soon becomes yellow and develops into prominent spots. *Strain A* virus has a marked effect in retarding the development of pollen grains, only 24 per cent of the grain from affected plants being of the normal size.

Host Range of Nicotiana Virus 12A. It is not known whether the host range is as great as that of the type virus, but it has been recorded by Valleau from the horse-nettle (*Solanum carolinense*) and the potato.

Distribution of Nicotiana Virus 12A. The yellow ringspot seems to be widely distributed in the State of Kentucky, U.S.A., where it has been recorded from five counties.

NICOTIANA VIRUS 12B

Synonym. *Green Ringspot Virus*, Valleau, 1932.

The Virus

Price (71) has shown that the type ringspot virus gives complete protection in an affected tobacco plant against the green ringspot virus (*Strain B*) and *vice-versâ*. This seems fair evidence that the two viruses are closely similar in nature.

Transmission. The virus is sap-transmissible, and, like *Strain A*, is carried in the seed of affected tobacco plants in a percentage of cases (104).

Diseases caused by Nicotiana Virus 12B

Nicotiana tabacum. Tobacco, var. Turkish. *Green ringspot*. The disease produced is somewhat similar to that caused by *Strain A*, the chlorotic line patterns developing in the same way, except that the lighter coloured tissue is light green instead of yellow. After the period of active development of necrotic rings, affected plants grow almost normally in contrast to plants affected with *Strain A*, which become chlorotic towards the tip. Affected seedlings from seed of tobacco plants infected with *Strain B* remain green and are usually nearly normal in appearance, rarely developing ringspot patterns.

Host Range of Nicotiana Virus 12B. The host range appears to be very similar to that of *Strain A*; it has been isolated from five potato plants of different origin, in which it produced apparently the same disease as *Strain A*. The virus has also been observed as one cause of cucumber "mosaic" in the State of Kentucky, U.S.A., where it is widely distributed.

CHAPTER V

Nicotiana Viruses 13-15 ; Lycopersicum Viruses 1-6 ; Hyoscyamus Virus 1 ; Datura Virus 1

NICOTIANA VIRUS 13. Price

Synonym. *Tobacco Ringspot No. 2*, Price, 1936.

The Virus

This virus was first isolated by Price (71) from affected Turkish tobacco plants in a glasshouse in Princeton, U.S.A.

Resistance to Alcohol. Infectivity is destroyed in twenty-four hours by 70 per cent ethyl alcohol, but not by 60 per cent alcohol during the same period.

Thermal Death-point. Inactivated by ten minutes' exposure in the water bath at 60° C., but not by ten minutes at 55° C.

Dilution End-point. This seems to be between 1 : 100 and 1 : 1,000.

Resistance to Ageing. Resists three days in extracted sap at room temperatures, but not four days.

Filterability. The writer has carried out a few ultra-filtration tests with the virus and finds that it passes a Gradocol membrane of 0·100 μ , average pore diameter, though the average particle diameter of the virus has not yet been measured. So far as they go, however, these tests suggest that the average particle diameter is not more than 0·05 μ . This is a lower filtration end-point than that of *Nicotiana Virus 12*, which is tentatively given as 0·150 μ , and which assigns to this virus an average particle diameter of not more than 0·075 μ (see p. 270).

Transmission. The virus is readily transmissible by rubbing, and Price (71) offers some evidence that it may be carried in a small percentage of the seed of infected Turkish tobacco plants.

Diseases caused by Nicotiana Virus 13

Nicotiana tabacum. Tobacco, var. Turkish. In tobacco the virus produces primary lesions consisting of zonate necrotic spots similar to, but distinct from, those produced by *Nicotiana Virus 12*. The virus likewise produces systemic lesions of the same type, and

plants affected systemically by it eventually recover in much the same manner as do plants affected with *Nicotiana Virus 12*. Frequently, however, the disease may become localised, in which case the virus fails to reach the young leaves and growing points. It is usually possible in such cases to induce systemic symptoms, and subsequent recovery, by cutting back the plants or by fertilising them heavily. Tobacco plants that have recovered from the disease caused by *Nicotiana Virus 13* are immune from a second attack by the same virus, but not from *Nicotiana Virus 12* (71) (see Fig 43, E).

Other Host Plants of Nicotiana Virus 13

The virus has been experimentally transmitted to the following species, on all of which the disease produced is characterised by the production of primary and systemic necrotic lesions.

Solanaceæ

Nicotiana glutinosa L., *N. langsdorffii* Weinm., *N. sylvestris* Spegaz. and Comes, *Lycopersicum esculentum* Mill.

Leguminosæ

Phaseolus vulgaris L., French or snap bean; *Vigna sinensis*, cowpea.

NICOTIANA VIRUS 14. Smith

Synonym. *Bergerac Ringspot Virus*, Smith, 1935.

The Virus

This virus was isolated by the writer in 1934. It was found in a tobacco plant growing at Bergerac in S.W. France. The plant in question was also infected with a distorting strain of *Nicotiana Virus 1*, and the ringspot virus was isolated by inoculation of the complex to bean (*Phaseolus*) in which *Nicotiana Virus 1* does not become systemic, while the ringspot virus does.

Resistance to Alcohol. Retains infectivity for twenty-four hours in 70 per cent alcohol, but not in 80 per cent alcohol.

Thermal Death-point. Inactivated by ten minutes' exposure at 80° C., but not by ten minutes at 75° C.

Dilution End-point. The virus is still infectious at a dilution of 1 : 100, but not at 1 : 1,000.

Resistance to Ageing. Resists for nine days at room tempera-

ture in expressed sap, but not for twelve days under the same conditions.

Diseases caused by *Nicotiana* Virus 14

Solanaceæ

Nicotiana tabacum, var. White Burley (see Fig. 43, B). Very thin necrotic rings develop on the rubbed leaves, five to seven days after inoculation ; these are followed by a general mottling with more or less incomplete chlorotic rings on the younger leaves. Sometimes a vein-banding effect develops with chlorotic rings or spots on the dark green islands. Recovery, so far as symptoms are concerned, is as complete with this virus as with the other two ringspot viruses (*Nicotiana Viruses 12 and 13*). For the diseases produced in other hosts, see below.

Comparison and Differentiation of the Three Ringspot Viruses (*Nicotiana Viruses 12, 13 and 14*)

In order to aid the identification and differentiation of these three very similar viruses, the following facts are given, derived from some unpublished work by Smith and d'Oliveira.

Physical Properties

Dilution End-point. *Nicotiana Virus 12* withstands a dilution of 1 : 1,000, but not 1 : 10,000. *No. 13 and No. 14* withstand 1 : 100, but not 1 : 1,000.

Thermal Death-point. For *Nos. 12 and 13* this is 60° C. for ten minutes, and for *No. 14* it is 80° C.

Resistance to Alcohol. *Nos. 12 and 13* are destroyed by twenty-four hours in 70 per cent ethyl alcohol, while *No. 14* is not.

Resistance to Ageing. *Nos. 12 and 13* resist for three days in extracted sap at room temperatures, but not for four. *No. 14* is still infectious after nine days under similar conditions.

Host Range. *Nicotiana tabacum* (White Burley). With *No. 12* a single necrotic ring surrounded by a chlorotic halo usually develops on the inoculated leaf. Systemic symptoms are very severe, producing necrosis of large areas with great reduction of the area of the leaf. With *No. 13* large circular necrotic lesions, often with a green centre, develop. These lesions are visible three to four days after inoculation, and four to five days later several concentric rings are formed. The systemic lesions are also necrotic. *No. 14* produces very thin necrotic rings on the rubbed

leaves. These are followed by a general mottling with more or less incomplete chlorotic rings on the younger leaves. Symptoms rapidly fade and the plants appear healthy while still retaining the virus (see Figs. 43, A, B and E).

N. glutinosa. No. 12 produces no local lesions as a rule on the inoculated leaves. Chlorotic rings, very small, concentric and sharply defined, develop later. No. 13 is apparently not transmissible to this plant. No. 14 produces local necrotic spots, small and roundish, five days after inoculation. Concentric rings may develop later outside these spots (see Fig. 43, C). Symptoms show systemically for a time with clearing of the veins and a slight crinkle of the whole plant.

Datura Stramonium. No. 12 produces depressed necrotic spots which penetrate through to the other side of the leaf. The local symptoms develop after five days, increase in size and coalesce. The whole plant then becomes strongly necrotic and is almost invariably killed (see Fig. 44, D). With No. 13 necrotic spots develop six days after inoculation; these spots are brown in colour with a well-marked centre. The affected leaves remain attached to the plant and its general growth is not affected. No. 14 gives rise to a very faint mottling with occasional small necrotic points. The plants are not affected in their general growth and all symptoms later disappear.

Lycopersicum esculentum. No. 12 produces no local symptoms on tomato and only a faint and transient mottling. Curiously enough, these symptoms seem much fainter than those described by Wingard as produced on tomato by grafting (p. 278). No. 13 produces no local lesions on tomato, but sometimes rings, which are brown and usually very faint, develop on the young leaves. More frequently the systemic symptoms are in the form of brown spots or incomplete rings which never become necrotic. There are no local lesions with No. 14, but nine to ten days after inoculation a faint mottling appears on the fully developed leaves, turning into a necrotic network three or four days later.

Phaseolus vulgaris, var. Canadian Wonder. No. 12 produces occasional necrotic rings on the inoculated leaves of the French bean. These rings later spread along the veins and involve the whole plant in a systemic necrosis which usually proves fatal within fifteen to twenty days. No. 13 produces numerous concentric rings on the inoculated leaves. The systemic lesions are brown, very small and abundant and sometimes cover the whole leaf. With No. 14 brown local lesions appear seven to nine

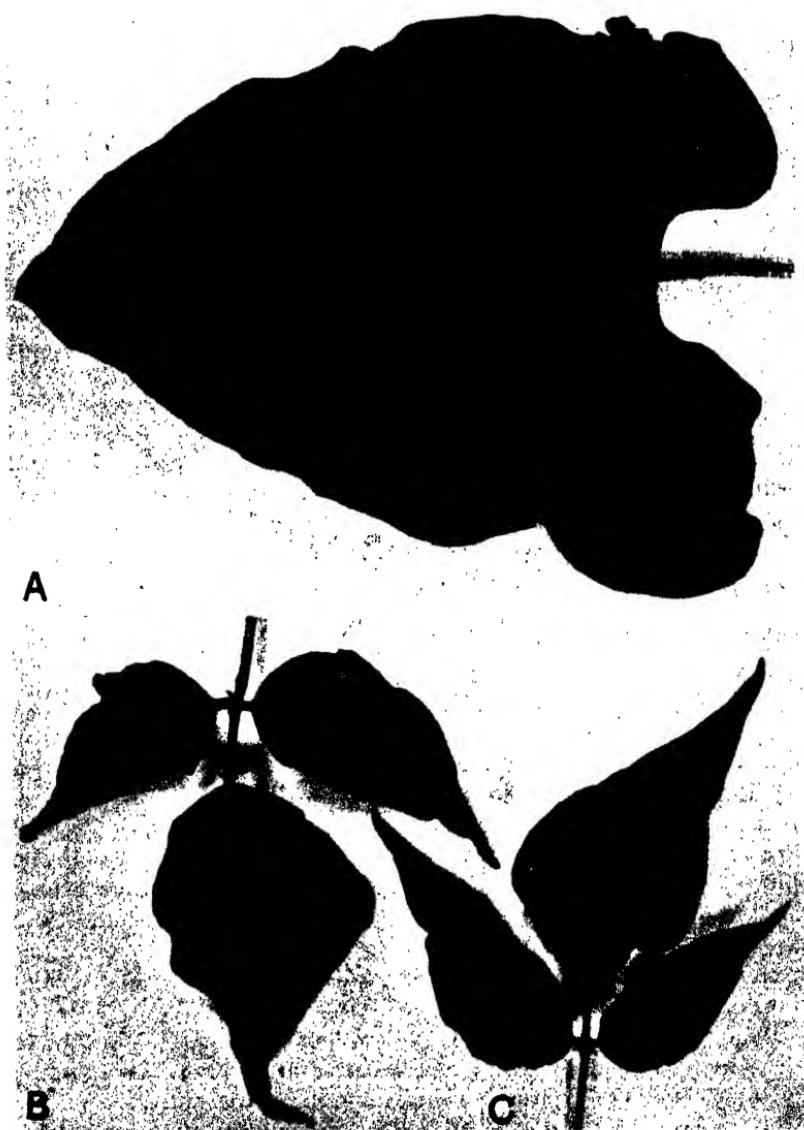


FIG. 45. Leaves of beans (*Phaseolus vulgaris*) infected with three different tobacco ringspot viruses.

- A. *Nicotiana Virus 12* (tobacco ringspot virus, No. 1, Price).
- B. *Nicotiana Virus 18* (tobacco ringspot virus, No. 2, Price).
- C. *Nicotiana Virus 14* (Bergerac ringspot virus).

days after inoculation. In the systemic infection numerous small necrotic points are produced, sometimes with a crinkle effect (see Fig. 45).

Differential Hosts

The differential hosts may be briefly summarised thus : No. 12 is distinguished from the other two by the fact that it kills *Datura Stramonium*, and further from No. 13 by the fact that the latter is not transmissible to *N. glutinosa*. No. 13 is distinguished from No. 14 by the production of necrosis on *Datura Stramonium* and also by its behaviour on tomato. From No. 12 it is distinguished by the fact that it is not transmissible to *N. glutinosa*. No. 14 is easily differentiated from the other two by its behaviour on tobacco (White Burley), by the complete absence of systemic necrosis, and by the production of numerous chlorotic rings on the non-inoculated leaves. No cross-immunity exists between these three ringspot viruses.

NICOTIANA VIRUS 15. Smith

This is a new virus, recently isolated by the writer, and it has not yet been described. It was found in the experimental glasshouses at Cambridge on a single tobacco plant. The virus is only feebly infectious and appears to be very slow-moving in the plant. In *Nicotiana glutinosa* under summer-time conditions, it produces a severely necrotic disease which is partially or wholly systemic (see Figs. 59, D and E). The leaves develop a necrotic patch which causes distortion and uneven growth of the rest of the leaf. Slight mottling may be present on the younger leaves, but the main symptom is necrosis. In winter time the virus becomes much less infective and is extremely hard to transmit. When transmission is effected, the amount of multiplication within the plant seems very small.

LYCOPERSICUM VIRUS 1. Bewley

Synonyms. *Tomato Stripe Virus*, *Tomato Streak Virus*, *Tomato Glasshouse Streak Virus*, Jarrett, 1930; *Single Virus Streak*, Ainsworth, Berkeley and Caldwell, 1934; *Tomato Virus 4*, J. Johnson's classification.

The Virus

*

Thermal Death-point. Survives exposure to 80° C. for ten minutes, but is destroyed by ten minutes' exposure to 90° C.

Longevity in vitro. Survives for several years in sterile extracted sap.

Desiccation. The virus is not destroyed by desiccation.

Photodynamic Action of Methylene Blue. After twenty minutes' exposure of a mixture of the virus and methylene blue to a 500-watt lamp the virus is slightly reduced in virulence (16).

Ultra-filtration. The filtration end-point of the virus as measured by Elford's Gradocol membranes is 0.086μ , giving an approximate particle diameter of 30 to $40\text{ m}\mu$ (82). The virus is not held back by Pasteur-Chamberland filters L_1 to L_8 .

Transmission. The virus is very infectious and is easily transmissible by sap-inoculation. The insect vectors, if any, are not known. Opinion appears to be divided on the subject of seed transmission of this virus. According to Berkeley and Madden (18) the virus is carried in the seed, but other workers (51) are of the opinion that this is not so.

Differential Hosts

Lycopersicum Virus 1 has several points in common with *Nicotiana Virus 1*, but it can be distinguished from the latter virus by the fact that it almost invariably produces local lesions on the tobacco plants which are either the only symptom or are followed by a severe systemic necrosis frequently accompanied by a mottle. *Nicotiana Virus 1* rarely produces necrotic local lesions on the inoculated leaves of the tobacco plant, though it sometimes gives rise to chlorotic spots. *Solanum melongena* also differentiates the two viruses in a similar manner (1).

Diseases caused by *Lycopersicum Virus 1*

Solanaceæ

Lycopersicum esculentum. Tomato. *Streak*. The streak disease of tomato caused by this virus is a serious one. Under commercial conditions of tomato culture in England the disease does not usually appear until about April, at the time of ripening of the first fruits, but it is occasionally seen in seedlings. The characteristic symptoms consist of necrotic lesions on stem, leaves and fruit. On the stems of affected plants the lesions take the form of dark longitudinal streaks which may be few and comparatively short, or numerous and elongated. Such stems are brittle and easily broken, and in the pith and cortex are brown areas. The leaves also show necrotic spots and patches, which

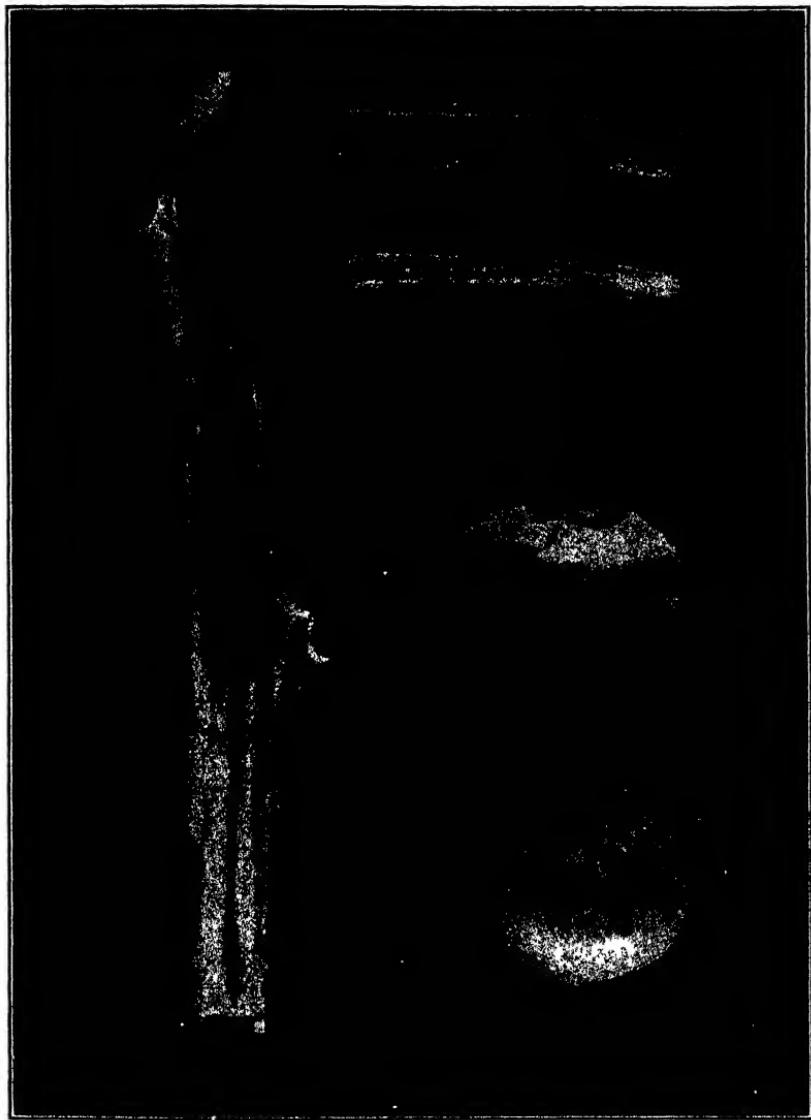


FIG. 46. *Lycopersicum Virus 1* (causing single virus streak).

(Left) Portion of an affected tomato stem showing the dark longitudinal streaks.

(Right, above) A short piece of stem cut open lengthwise; the upper portion shows the browned shrunken internal tissues ; the lower the external streaks.

(Right, below) Two affected fruits.

(After Bewley and Paine.)

subsequently enlarge, causing the leaves to shrivel. On affected fruits rounded or irregular sunken blotches may occur (Fig. 46).

Alternatively, the virus may produce in tomato plants a mottling disease only, without necrosis. Such a disease resembles very closely the ordinary tomato mosaic (see p. 244). The precise environmental conditions governing the development of this alternative type of symptom are not known.

Nicotiana tabacum. Tobacco. Necrotic local lesions develop on the inoculated leaves three or four days after inoculation. These lesions enlarge somewhat and may be the only symptom, or alternatively they sometimes coalesce. In this case the necrosis spreads along the petiole and enters the stem in which a severe longitudinal lesion develops. Plants affected in this way usually collapse and may be killed. If they survive, however, growth recommences from axillary buds low down on the stem. Such secondary growth may show mottling symptoms only.

On certain other Solanaceous plants the virus produces mottling symptoms similar to those produced on the same plants by *Nicotiana Virus 1*. These are *Nicotiana glutinosa*, *Datura Stramonium*, *Solanum ciliatum*, *Physalis pubescens*, and *Capsicum annuum* (1).

Geographical Distribution. The virus occurs commonly in the commercial tomato crops in the British Isles. It has been isolated from diseased tomatoes sent from Canada (3). The virus may also be found in the U.S.A., but there is little information on this point. The most common tomato streak disease in that country is the composite one caused by infection with two viruses (see p. 330).

Control Measures. Since this virus is of the infectious type which can be carried on the pruning knife or hands, cultural operations should not be carried out on diseased and healthy plants at the same time. A single diseased plant is sufficient for the virus to be spread through the whole crop under commercial glasshouse conditions. Healthy plants should be dealt with first and the knife carefully disinfected by dipping in some antiseptic such as a 2 per cent solution of Lysol. Where nitrogenous manure has been given in excess, soft growth has been made and the streak disease has appeared, top dressing the soil with sulphate of potash at the rate of 5 to 10 cwt. per acre is recommended. Half the amount should be given immediately the symptoms of streak appear and the other half about a fortnight later. It is unwise to save seed from affected tomato plants.

LYCOPERSICUM VIRUS 1A

A yellow strain of *Lycopersicum Virus 1* has been isolated by the writer (82). Its general characteristics appear to be similar to the type virus, but it produces no necrotic symptoms. It gives rise to local *chlorotic* spots on the inoculated leaves of tobacco plants, and these are followed by a yellow systemic mottle. The virus appears unable to withstand the same degree of dilution as the type virus, and the two strains can be separated by ultra-filtration.

LYCOPERSICUM VIRUS 2. Valleau and Johnson

Synonyms. *Tomato Ring Mosaic Virus*, Valleau and Johnson, 1930; *Ring Mosaic Streak Virus*, Ainsworth *et al.*, 1935.

The Virus

Thermal Death-point. The virus survives ten minutes' exposure to 70° C., but is destroyed by ten minutes' exposure to 80° C.

Resistance to Ageing. Survives some years in dried tissues.

Filterability. Passes Pasteur-Chamberland filters L₁ to L₇ (3).

Transmission. The virus is sap-transmissible, but there is no information on the question of seed transmission and insect vectors.

Differential Host

On tobacco, Burley variety, this virus produces mottling and distortion somewhat like *Nicotiana Virus 1*, but it can be distinguished by the production, in addition, of definite rings consisting of alternate bands of pale green to yellow tissue. In greenhouse plants these rings are usually almost perfect circles (46). The virus is not transmissible to cucumber.

Diseases caused by *Lycopersicum Virus 2***Solanaceæ**

Lycopersicum esculentum. Tomato. *Ring Mosaic*. According to Johnson (46) this virus produces very striking symptoms on tomato. Circular, water-soaked necrotic spots, $\frac{1}{4}$ inch in diameter, develop on the rubbed leaves seven to ten days after inoculation. Necrotic streaks about $\frac{1}{2}$ to $\frac{1}{4}$ inch long develop on the mid-veins of these leaves a few days later. Leaflets affected in this way usually droop markedly. The necrosis gradually spreads from the mid-veins to the lamina, the leaflet eventually dying. Streaks gradually form in the main axis of the leaf and then in the stem.



A



C



D



Yellow areas may occur as oakleaf patterns, and concentric yellow patterns sometimes develop between the veins. Concentric sunken, chlorotic rings, $\frac{1}{8}$ to $\frac{3}{8}$ inch in diameter, appear on the fruit when about half-grown.

Under English glasshouse conditions the disease caused by this virus seems less severe in the foliage of affected tomatoes. There are occasional yellow areas on the leaves, with little mottle or distortion, while necrotic lesions are irregularly distributed on stem, petioles and leaves of older parts of the plant (see Fig. 47, B). There is little stunting. The fruit is more severely affected and green fruits exhibit chlorotic or necrotic areas and ripe fruit is disfigured by the irregular development of red pigment in the skin (3).

Nicotiana tabacum. Burley variety. Local lesions develop on the rubbed leaves and these are followed by a systemic chlorosis. Rings which may become necrotic appear about a week later. Under greenhouse conditions these rings take the form of complete circles, but in the field they appear more like necrotic spots (46).

N. glutinosa. Local necrotic lesions, only, develop on the rubbed leaves of this plant. There is no systemic infection.

Capsicum annuum. Bell pepper. Affected plants have local spotting, but the mottling is obscure. Ring-like patterns composed of alternate bands of green and yellow tissue appear on the rubbed leaves, such leaves eventually turn yellow. Affected pepper plants are severely distorted and dwarfed (46).

Datura Stramonium L. Jimson weed. Necrotic local lesions develop on the inoculated leaves, and these are followed by necrosis of the mid-ribs and petioles. The inoculated leaves finally shrivel or fall. Systemic infection rarely develops in this species.

Geographical Distribution. *Lycopersicum Virus 2* has only been recorded from Kentucky in the U.S.A.

FIG. 47.

- A. Tomato mosaic due to infection with *Nicotiana Virus 1* (tobacco mosaic virus).
- B. Tomato plant infected with *Lycopersicum Virus 2* causing ring mosaic streak.
- C. Fruits of tomato plant infected with *Nicotiana Virus 1C* causing tomato aucuba mosaic.
- D. Fruits of tomato plant infected with *Lycopersicum Virus 8*, tomato spotted wilt virus.
(After Ainsworth, Berkeley and Caldwell.)

LYCOPERSICUM VIRUS 3. Brittlebank

Synonyms. *Tomato Spotted Wilt Virus*, Brittlebank; *T.S.W. Virus*; *Kromnek or Kat River Disease Virus*, Moore; *Tomato Virus 1*, J. Johnson's classification; possibly *Pineapple Yellow-spot Virus*, Linford.

The Virus

Reactions with Chemicals. The virus is rapidly inactivated *in vitro* by 0·001 M solutions of oxidising agents which induce in the suspensions a potential greater than 0·2 volt at pH 7. KCN, in 0·01 M solution, protects the virus both against anaerobic and aerobic inactivation. $HgCl_2$, in 0·001 M solution, causes instantaneous inactivation of the virus. Cathecol, quinol and phenol alone inactivate the virus in the presence of air, but do not do so if Na_2SO_3 is also present. It is probable that secondary oxidation products cause this inactivation (13A).

Thermal Death-point. The virus is destroyed by quite low temperatures, the thermal death-point for ten minutes' exposure being close to 42° C.

Dilution End-point. Under optimum conditions the virus will stand a dilution of between 1 : 10,000 and 1 : 100,000.

Resistance to Ageing. The virus is rapidly inactivated in expressed sap, and loses infectivity in less than five hours at room temperatures. This rapid inactivation is probably due to oxidation effects, and the longevity of the virus can be increased by keeping it at low temperatures or by the addition to the sap of certain reducing agents such as sodium sulphite (7). This effect of sodium sulphite has a practical application in aiding diagnosis of the disease caused by the virus in certain plants where inactivation of the agent in expressed sap is unusually rapid (see Chrysanthemum, p. 302).

Desiccation. The virus is destroyed by drying.

Filterability. All attempts to filter this virus through Pasteur-Chamberland candles have up to the present proved negative. This failure is probably due, at least in part, to the rapid loss in potency of the virus during the filtration process. The virus has been filtered through a Gradocol membrane of approximate pore diameter $0\cdot45\mu$, but here again great difficulty was experienced in obtaining positive results.

Transmission. The virus is sap-transmissible, but certain conditions must be observed in order to obtain a high percentage of successful transfers. The source of inoculum should, if possible,

be a young recently infected plant, and the best method of inoculation is a gentle rubbing or wiping with a piece of muslin or a glass spatula dipped in the inoculum. The rubbing should be just sufficient to break the hairs of the leaf without injuring the mesophyll tissue. In transmitting the virus to some plants, such as the lettuce, for example, the addition to the inoculum of an abrasive, like carborundum powder, facilitates the transfer of the virus. Similarly it is advisable to add a small quantity of some reducing agent like sodium sulphite to the extracted sap of chrysanthemums when these plants are being tested for the presence of the virus (2).

Seed Transmission. It is highly improbable that *Lycopersicum Virus 3* is carried in the seed; what evidence there is suggests that the virus is not seed transmitted. The writer has grown large numbers of tomato plants from seed derived from infected tomato fruits and all the plants thus grown have proved to be free of the virus.

Insect Transmission. The insect vectors of the virus are several species of thrips. In the British Isles the species concerned is *Thrips tabaci* L., the onion thrips (see p. 462), and in Australia this species and *Frankliniella insularis* Franklin, the black carnation thrips (see p. 460). In California the vectors are stated to be *Thrips tabaci* L., *Frankliniella occidentalis* Perg. and *F. moultoni* Hood (23). It is quite possible that in other countries, in South Africa, for example, still other species of thrips may transmit the virus.

There are two interesting facts connected with the transmission of the virus by thrips; the first is the inability of the adult insect to pick up the virus *de novo*. It is necessary for the insect to feed in the *larval* state upon an infected plant for it to become viruliferous. The adult arising from an infective larva is then itself infective (6). The second fact is the long delay in the development of infective power within the thrips; this period may be as long as nine days, while the shortest period is between five and six days.

Differential Hosts

Petunia sp. The petunia, garden variety, is an excellent indicator plant for this virus. Local lesions develop on the inoculated leaves within forty-eight to seventy-two hours. The lesions consist of circular spots with a reddish-brown margin and a paler centre. The virus rarely becomes systemic in the petunia.

Since *Nicotiana glutinosa* is exceedingly sensitive to infection, this plant is a useful indicator. Local lesions are produced in three to four days, and these gradually increase in size, forming spots of concentric necrotic zones about 2 to 3 mm. in diameter. These are usually, but not always, followed by a lethal systemic necrosis (see Fig. 52, B).

Diseases caused by Lycopersicum Virus 3

The virus of spotted wilt has an exceedingly wide host range and is one of the few viruses which will pass from dicotyledons to monocotyledons. It is not practicable to describe all diseases caused by this virus, and therefore a selection has been made of those which are more important and which commonly occur. A list of susceptible plants which is as complete as possible will be found on p. 311.

Dicotyledons. Ranunculaceæ

Delphinium sp. Affected plants are rather stunted and the leaves are somewhat distorted. Numerous distinct double concentric rings are sometimes present. The young leaves are malformed with the edges yellowish, necrotic and inwardly curled. Necrotic patches may develop on the stems and older leaves.

Papaveraceæ

Papaver nudicaule L. Iceland poppy. The first symptom is a clearing of the veins of the youngest leaves. These leaves become somewhat twisted, cease growth with the appearance of symptoms and gradually turn yellow. The whole centre of the plant acquires a pallid, stunted appearance. In affected field plants, where the rosette of leaves is usually more vigorously developed than in pot plants, a bunched habit is acquired due to the stunting and yellowing of many central leaves. Frequently purplish blotches appear on some of the yellowed leaves or on their mid-ribs or petioles, and a necrosis may set in which destroys the petiole while leaving the leaf blade unaffected. Although diseased plants usually remain alive for weeks in a stunted condition, a rapid extension of the necrosis seen on the petioles sometimes caused the sudden death of the plant. The flowering stems of affected poppies are frequently much twisted and bent over; when they do grow erect they are shorter than normal, more sappy and brittle, and the buds frequently open badly. The opening of the bud may be delayed until after erection

from the recurved position, so that the tip of the bud points vertically upwards, a condition not seen in healthy poppies. The sepals, instead of lifting normally from the receptacle, often confine the petals so long that they burst out laterally. On the flowering stems of many affected plants water-soaked areas may appear from which a drop of milky-white latex exudes. This soon becomes brown on contact with the air and dries to a small hard pellet. The exuding of a brownish drop of liquid from necrotic patches on the leaf petioles or mid-ribs in their early stages also occurs at times (6).

Plantaginaceæ

Plantago major Linn. Greater plantain. This very common weed is susceptible to infection, and this fact is of importance, since the weed may act as a source of contamination to nearby cultivated plants. Necrotic rings or spots appear on the leaves of affected plantains, together with some necrosis of the petiole.

Polygonaceæ

Polygonum convolvulus Linn. Black bindweed. This climbing weed has been observed by the writer affected with the virus out-of-doors. The most characteristic symptom is the development of numerous double or treble concentric rings upon the leaves.

Tropæolaceæ

Tropæolum majus L. Nasturtium. Symptoms consist of clearing of the veins of the youngest leaves at the time of infection ; this is followed by distortion and cupping of some leaves and the gradual development of an irregular pallidity with yellowish spots and mottling as the leaves become older. The plants are usually stunted. A characteristic symptom of the disease is the development of numerous pale necrotic spots which may cover the whole leaf surface (see Fig. 48, B).

Begoniaceæ.

Begonia sp. In affected begonias, rings or zoned spots develop on the leaves. There may be some mottling, the plants are stunted and the flowers are poor.

Leguminosæ

Phaseolus vulgaris. French bean, snap bean. The writer has experienced some difficulty in transmitting the virus to this plant, and it is not certain whether the species is susceptible or not.

Vigna sinensis. Cowpea. This plant is susceptible to infection, but symptoms do not appear to be very marked. Reddish lesions, usually circular or of a zoned type, develop on the leaves.

Vicia faba. Broad bean. The broad bean can be experimentally infected with *Lycopersicum Virus 3*, although natural infections seem to be rare, in England at least. Symptoms appear to be only necrotic, and lesions of a zoned type develop on the leaves, which rapidly shrivel and die. A characteristic of this disease is a lesion running longitudinally along the stem. Affected plants are usually killed.

Lupinus leucophyllus. Lupin. The effect of the virus on lupins is shown chiefly on the leaves, the symptoms consisting of necrotic concentric rings or circular spots. These necrotic spots give rise to a certain amount of distortion and deformity in the leaves (see Fig. 51, B).

Pisum sativum L. Garden pea. First symptoms appear seven to twenty days after infection as necrotic streaks on the stem and as spot or vein necrosis on young leaves. Frequently, however, the initial symptom appears as a distinct necrotic spot on the artificially inoculated leaves, or in association with thrips-feeding injury when the virus is transmitted by this insect. There is usually a streak on the stem, purplish or bluish-brown in colour, which may extend only a little beyond the point of inception or over the entire length of the stem. Histological examination of infected stems shows general necrosis of the parenchymatous cells, including the phloem. Unilateral development of affected parts is frequent. Occasionally a mottling pattern develops on leaves infected when young. Local infections of immature pods may appear as small circular necrotic spots associated with the thrips-feeding marks, and necrosis may later extend into irregular, wavy concentric patterns over most of the surface. Systemic infection may result also in necrotic or concentric patterns on the pods; if, however, these are systemically infected when very young, they often become stunted, distorted and collapsed. The seeds occasionally show necrotic spots or patterns (108).

The sweet pea, *Lathyrus odoratus*, is also susceptible to infection.

Compositæ

A great number of species belonging to this family are frequently affected with *Lycopersicum Virus 3*, among them being many well-known ornamental plants.

Callistephus chinensis. Aster. China aster. In affected

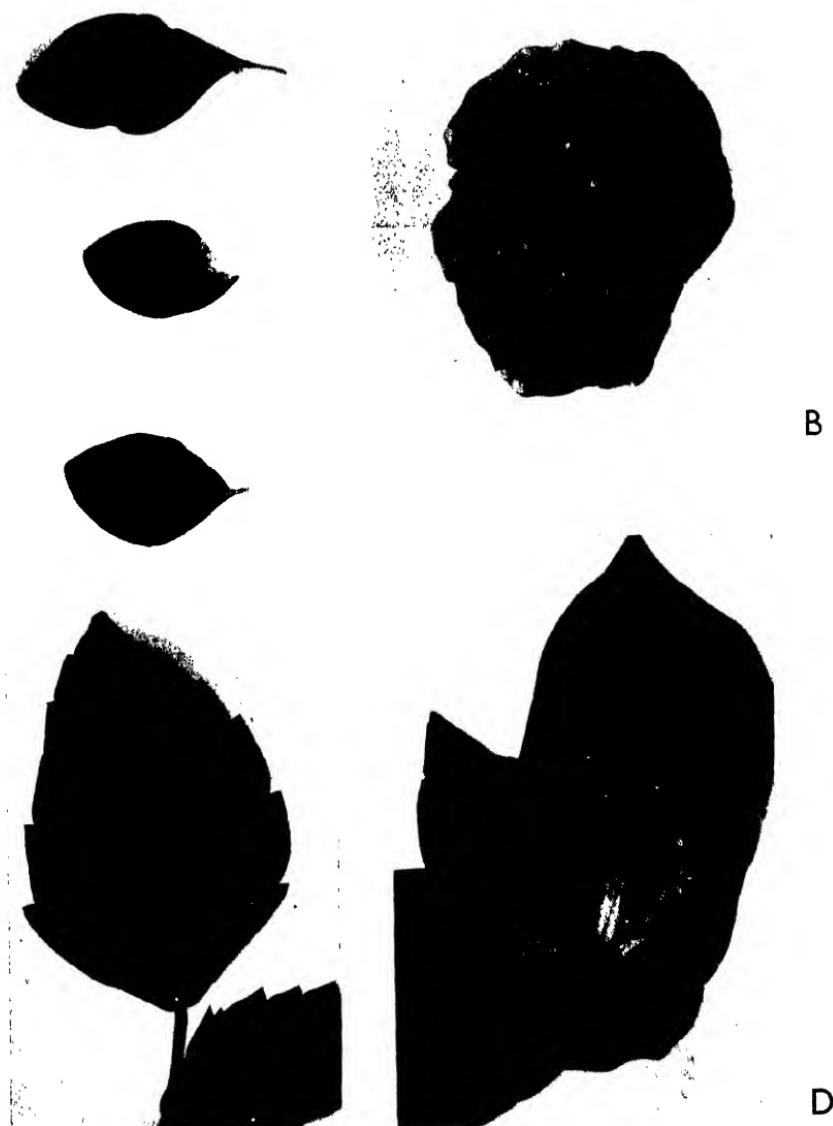


FIG. 48. *Lycopersicum Virus 3* (tomato spotted wilt virus).

- A. Leaves of *Streptosolen Jamesonii* with ring and line markings.
- B. Leaf of *Tropaeolum* (nasturtium) with numerous necrotic spots.
- C. Leaf of infected *Dahlia* showing ring and line markings.
- D. Leaf of infected *Zinnia*.

asters the central leaves are distorted, giving the plant a rosetted habit of growth. There is usually a dark green mottling or streaking on the leaves and flower bracts. The flowers are abnormal in appearance, very small and frequently much distorted. Ring-formation, so characteristic of this virus, has not been observed on this species.

Chrysanthemum sp. The chrysanthemum is an important host plant, partly because it so frequently follows after the tomato crop in English glasshouses and partly because it offers the virus good opportunity for overwintering under English conditions. In young chrysanthemum plants and cuttings the first sign of infection consists of pale chlorotic areas on the upper leaves, followed by the most characteristic symptom, a bronzing of the leaves somewhat similar to that produced on the tomato by the same virus (see p. 305). This is rapidly followed by a rather severe necrosis of the leaves and sometimes of the stem. Young plants are frequently killed. In older affected plants the bronzing or "rustiness" is also present, and there may be a slight mottling of the leaves. Growth is poor and affected plants are stunted as compared with the normal. The virus is inactivated almost instantaneously in the expressed sap of chrysanthemum, and it has been shown (2) that the addition of 0·5 per cent solution of anhydrous sodium sulphite to the extracted sap retards the inactivation and is thus a valuable aid in the diagnosis of this disease in chrysanthemums (see Fig. 12, A).

Cineraria sp. This species is very susceptible to infection and the resulting disease is severe. Pale yellow spots develop on the leaves, and these are followed by browning of the veins and frequently by the death of the plant.

Dahlia sp. The dahlia plant is very commonly infected with *Lycopersicum Virus 3* (spotted wilt virus). The symptoms are of two general types, a well-defined mosaic mottle and/or the production of concentric rings or wavy lines on the leaves. The disease may be severe on young seedlings, but is usually rather mild on older plants. The dahlia is an important host not only because the disease is propagated year after year, but also because the virus is transported to new districts in the tubers (see Fig. 48, C).

Zinnia elegans. The zinnia is another ornamental plant frequently infected with *Lycopersicum Virus 3*. The leaves of affected plants show a well-marked mosaic mottling (see Fig. 48, D). The mottling may consist of numerous flecks of dark green on a light green surface or alternatively of chlorotic rings of a light



FIG. 49. *Lycopersicum Virus 3* (tomato spotted wilt virus).
A and B. Affected Romaine lettuce, showing necrotic spots and curvature of
the leaves.
C. Advanced symptoms ; necrosis of lamina and veins.
(After Tompkins.)

colour on a darker background. The growing points of affected plants are sometimes twisted and distorted.

Lactuca sativa, var. *capitata* L. Head lettuce. *Lactuca sativa*, var. *longifolia* Lam. Romaine and cos lettuce. Infected plants of lettuce are unmarketable and frequently die prematurely. The disease causes a slight marginal wilting, necrotic spotting and slight yellowing of the leaves, usually on one side of the plant, with consequent tilting of the head towards the affected side. Lateral curvature of many leaves is produced. The necrotic spotting involves even the youngest leaves, and in older plants difficulty is experienced in detecting the internal necrosis. Lettuces may be infected at any age (103). The heads of diseased lettuces always lean to one side in contrast to erect healthy heads. In the field, infection is first observed in the form of necrotic spotting on the upper part of one of the young inner leaves (see Fig. 49).

Primulaceæ

Primula malacoides. Symptoms consist of yellowing of the leaves, which eventually wither, and marked stunting. Necrosis does not seem to be a characteristic of the disease in this species, and lesions do not form on leaves artificially inoculated. Infected plants rarely flower and the disease is usually fatal.

Primula sinensis. Irregular necrotic lesions develop on the leaves frequently near the edges. These lesions fuse together, forming large scorch-like patches which result in the death of several leaves. The veins of the younger leaves become prominent and slightly yellow. The whole plant is stunted and infected plants seldom flower. In contradistinction to *P. malacoides*, large necrotic lesions develop on leaves artificially inoculated.

Campanulaceæ

Campanula pyramidalis and *Trachelium* sp. show similar symptoms when infected with *Lycopersicum Virus 3*. The general trend of the disease is towards ringspot formation on the leaves, usually accompanied by wavy lines and markings. Affected plants are stunted and growth is poor (see Fig. 51, C).

Solanaceæ

Lycopersicum esculentum. Tomato. *Spotted Wilt*. It is from the disease it causes in the tomato that *Lycopersicum Virus 3* gets its popular name of spotted or bronzy wilt (see Fig. 50).

The symptoms on the tomato plant are characteristic and easily

recognised. The initial signs of the disease appear as a slight intensification or thickening of the veins of the youngest leaves ; this is sometimes accompanied by one or two concentric rings. At about the same time the young leaves exhibit a tendency to curl slightly downwards and inwards. The next symptom is the appearance of a very characteristic bronzing of the leaves. This bronzing may cover the leaf surface completely, or, as is more usual in glasshouse plants, may be in the form of bronze coloured circular markings. At this stage the plant is stunted in its growth as compared with normal plants. Occasionally affected plants are killed outright by the bronzing, which develops into a severe necrosis ; this usually occurs when the plant is infected as a young seedling. Normally, however, the plant is not killed. At a later stage of the disease symptoms may take the form of a fairly bold yellowish mosaic mottling of the leaves, together with some leaf distortion.

The fruit of affected tomato plants may show no symptoms, particularly if the fruit trusses have been formed previous to infection. On the other hand, pronounced symptoms may appear on the fruit. They consist usually of paler red, often yellow, or more rarely white areas in the normal red skin of the ripe tomato. These pale areas are of the most varied shapes, ranging from an irregular mottling or blotchiness to very distinct concentric circles. Sometimes practically the whole of the skin is yellowish, with only small, sharply defined islands where the normal red colour develops (74, 79) (see Fig. 47, D).

Nicotiana tabacum. Tobacco, White Burley, Virginia. Local lesions develop on the inoculated or initially infected leaves of tobacco. These may be either in the form of concentric rings with a central spot, or, more usually, consist of large plaque-like lesions composed of concentric zones of necrotic tissue. The progress of systemic infection varies according to environmental conditions ; further development of concentric necrotic rings may occur on the young leaves and, more rarely, on the stem. In some cases the leaf surface may be almost entirely covered with small necrotic rings (see Fig. 56, C). Another form of the disease in tobacco consists of a systemic and usually fatal necrosis. In this type of symptom rings seldom develop, and the initial necrosis is usually confined to the veins which become yellow and strongly outlined. From this stage the progress of the disease is rapid. The leaves quickly lose their normal green colour and turn dark grey with a scorched appearance. At this point the plant either

dies completely or dies down except for the central shoot. In the latter case, growth is resumed after a period, and an apparently healthy plant is produced which may grow normally for a week or two, after which the necrosis appears again. Occasionally tobacco plants of long-standing infection may show a slight mottling of the leaves. The writer has also observed cases where infection was confined to the inoculated leaves and the virus did not move out of the local lesions, presumably because it was cut off from further multiplication by the rapid spread of necrosis.

Nicotiana glutinosa. This species is very susceptible, initial symptoms appearing as small circular necrotic spots four or five days after infection. As a rule these spots are fewer in number, but become much larger than the local lesions produced by *Nicotiana Virus 1* on this plant (see Fig. 52, B). Systemic symptoms appear after some seven to ten days, and usually consist of a sudden necrosis along the fine veins in the basal portions of the youngest leaves. The plant ceases growth; the centre leaves become pallid, with the margins bending downwards, and necrosis gradually progresses until the whole plant is dead, a process which may be complete in a fortnight or which may take several weeks.

Nicotiana acuminata. In this species the local lesions are less necrotic and are more of the "yellow blotch" type. These are followed by clearing or pallidity of the tissue in the basal portions of the young leaves, the affected tissue then becoming necrotic.

Solanum capsicastrum. Winter Cherry, Orange Flower, Star Capsicum. The spotted wilt virus produces symptoms of the ringspot type and the ring formation is shown at its highest development in this species. Frequently the leaves of affected plants are covered with concentric circles, the numbers of such concentric rings in each group varying from two or three to seven or eight. A certain amount of necrosis in the region of the veins is also sometimes present. Affected plants are smaller than normal. *S. capsicastrum* is an important host, since it enables the virus to overwinter in English glasshouses, and being propagated largely from cuttings it is also instrumental in spreading the virus from one locality to another.

Solanum seaforthianum. This species is usually killed outright. The secondary growth symptoms consist of rather distorted leaves in which light green and dark green areas are frequently present, though not, as a rule, in a typical mosaic manner.

Petunia hybrida. *Petunia* spp. Garden varieties. In Petunia



FIG. 51. *Lycopersicum* Virus 3 (tomato spotted wilt virus)

- A. Symptoms on infected *Calceolaria*.
- B. Leaf of infected lupin.
- C. Leaf of infected plant of *Trachelium* sp., showing rings.

local necrotic lesions are formed, and in several of the garden varieties no further development of the disease ensues. These lesions are ring-like with a brown margin, later the centre becomes lighter, the margins remaining dark.

Streptosolen Jamesonii. *Browallia speciosa major*. These two species are fairly frequently infected with the spotted wilt virus. On both plants the symptoms take the form of rings or concentric circles on the leaves with a varying amount of necrosis (see Fig. 48, A).

Datura Stramonium. Jimson weed. Rather characteristic symptoms develop on this species; these may take the form of concentric rings or, alternatively, of a vein necrosis producing a type of oak-leaf pattern on the leaf.

Among other Solanaceous hosts susceptible to this virus are *Capsicum annuum*, *Hyocymus niger*, *Lycium ferocissimum*, *Physalis peruviana*, *Salpiglossis* sp. and *Schizanthus* sp.

The general course of the disease in these species may be summarised as follows:—

(1) After two to fifteen days the development of local lesions on the inoculated leaves; (2) after eight to twenty days the development of general symptoms at the growing point, involving cessation of growth, with or without varying degrees of necrosis on the developing leaves; (3) after some two to three weeks at a standstill the gradual production of a rather poor, under-developed, frequently malformed secondary growth (6).

Scrophulariaceæ

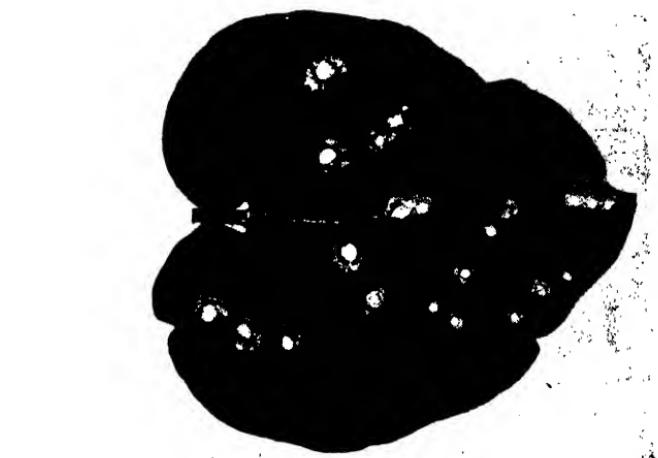
Calceolaria sp. The calceolaria is very susceptible to the virus, and this plant and its varieties are frequently infected under English glasshouse conditions. The symptoms are characteristic and severe. Diseased plants, particularly if long infected, are stunted with the growing points tending towards rosette formation. The most outstanding character, however, is the presence of large pale irregular blotches on the leaves which give a curious patchwork effect to the plant. The leaf outline is also distorted and the green tissue between the blotches may be raised in blisters. Frequently accompanying these symptoms is a red or pinkish necrosis of the tissues in the neighbourhood of the veins (see Fig. 51, A).

Gesneriaceæ

Gloxinia sp. Very characteristic symptoms are produced by this virus on gloxinia. Large single rings with wide dark brown



C



B



A

FIG. 52. *Lycopersicum Virus 3* (tomato spotted wilt virus).

- A. Leaf of infected *Gloriosa*, natural infection.
- B. Leaf of *Nicotiana glutinosa*, showing local lesions caused by this virus.
- C. Systemic infection of potato, var. Arran Victory, experimental infection.
(B, after Samuel and Bald.)

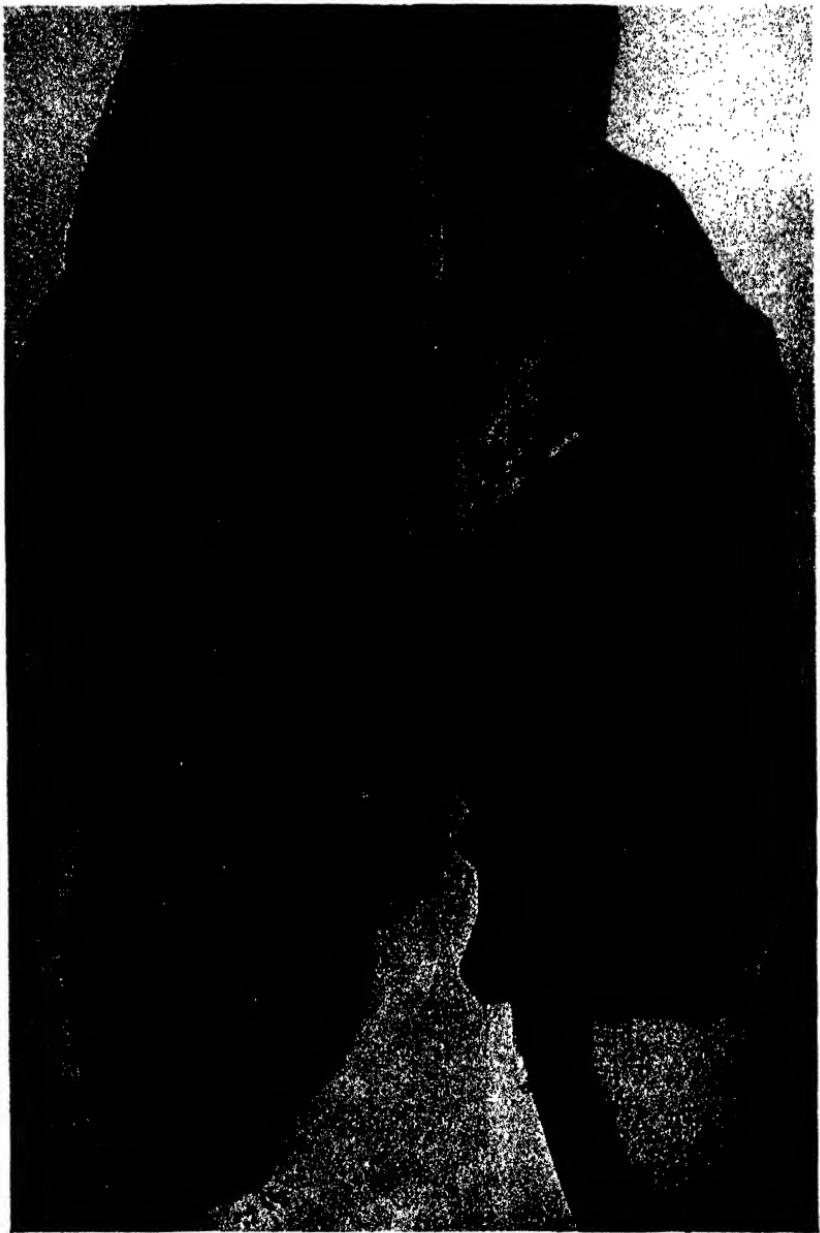


FIG. 58. *Lycopersicum Virus* 8 (tomato spotted wilt virus).
Symptoms on arum lily (*Richardia africana* Kunth.). Note the characteristic
circular white spots in the neighbourhood of the veins.

walls develop on the leaves. These rings may be isolated or there may be several close together; they are not as a rule concentric (see Fig. 52, A).

Monocotyledons. Araceæ

Richardia africana Kunth. (*Zantedeschia aethiopica*). Arum or calla lily. Under English glasshouse conditions arum lilies are very frequently infected with the spotted wilt virus, partly because of the fondness of the thrips for the flowers of this plant and partly because arums frequently follow the tomato crop in the same glasshouse. The symptoms take the form of numerous whitish spots, often ring-like in character, on the leaves, usually in the neighbourhood of the veins (see Fig. 53). They may be sufficiently numerous to cover the leaf. In addition, white streaks develop on the leaf and flower stalks and pale blotchy spots on the green flower buds. The leaves are crinkled and twisted and the flowers deformed.

Amaryllidaceæ

Hippeastrum sp. The leaves of affected plants show numerous pale yellow or white spots. These spots may be isolated or coalesced into pale patches. Accompanying them are numerous blood-red necrotic spots usually associated with the pale spots, but also running along and destroying the leaf edges. The leaves finally turn yellow and die.

Host Range of Lycopersicum Virus 3 (Spotted Wilt Virus).

Dicotyledons

Ranunculaceæ

Delphinium sp., *Paeonia* sp., *Ranunculus* sp., *Anemone* sp., *Aquilegia vulgaris*.

Papaveraceæ

Papaver sp. *P. nudicaule*.

Cruciferæ

Brassica oleracea, var. *botrytis*. L. cauliflower, *Cheiranthus* sp.

Polygonaceæ

Polygonum sp. Bindweed.

Tropæolaceæ

Tropæolum sp. Nasturtium; *Tropæolum majus*.

Begoniaceæ

Begonia sp.

Leguminosæ

Lupinus sp. and *Lupinus leucophyllus*. Lupin. *Vicia faba*. Broad bean. *Vigna sinensis*. Cowpea. *Phaseolus vulgaris*. French or snap bean. *Pisum sativum*. Garden pea.

Umbelliferæ

Apium graveolens. Celery.

Dipsacaceæ

Scabiosa sp.

Compositæ

Dahlia variabilis, *Callistephus chinensis*, China aster, *Aster* sp., *Zinnia* sp., *Zinnia elegans*, *Chrysanthemum* sp., *Cineraria* sp., *Lactuca sativa*, var. *capitata*, *Lactuca sativa*, var. *longiflora* Lam., Romaine and cos lettuce, *Emilia* sp., *Gaillardia* sp., *Layia* sp., *Coreopsis (Calliopsis) drummondii*, *Cosmos* sp., *Calendula officinalis*.

Primulaceæ

Primula sp., *P. malacoides*, *P. obconica*, *P. sinensis*.

Campanulaceæ

Campanula sp., *C. pyramidalis*, *Trachelium* sp., *T. cæruleum*.

Lobeliaceæ

Lobelia sp.

Solanaceæ

Lycopersicum esculentum. Tomato.

L. pimpinellifolium. Small red-current tomato.

Nicotiana acuminata.

N. alata.

N. angustifolia.

N. atropurpureum.

N. bigelovii.

N. calyciflora.

N. caudigera.
N. chinensis.
N. glauca.
N. glutinosa.
N. langsdorffii.
N. longiflora.
N. macrophylla.
N. paniculata.
N. rustica.
N. sanderæ.
N. suaveolens.
N. sylvestris.
N. tabacum. Tobacco.
Solanum aculeatissimum.
S. Capsicastrum.
S. Dulcamara. Woody nightshade.
S. laciniatum.
S. marginatum.
S. Melongena L. Egg plant.
S. miniatum.
S. nigrum. Black nightshade.
S. nodiflorum.
S. sanitwongsii.
S. seaforthianum.
S. sodomaeum.
S. tuberosum. Potato.
Datura Stramonium.
D. Wrightii.
Capsicum sp.
Capsicum annuum.
Petunia sp.
Petunia hybrida.
Streptosolen Jamesonii.
Browallia speciosa-major.
Hyoscyamus niger L. Henbane.
Atropa belladonna. Deadly nightshade.
Physalis peruviana L. Cape gooseberry.
Lycium ferocissimum.
Salpiglossis sp.
Schizanthus sp.
Physalis sp.
Nicandra sp.

Scrophulariaceæ

Calceolaria sp.
Antirrhinum sp.
Penstemon sp.

Gesneriaceæ

Gloxinia sp.

Labiatae

Salvia sp.

Monocotyledons. Araceæ

Richardia africana Kunth. (*Zantedeschia aethiopica*). Arum lily.

Amaryllidaceæ

Hippeastrum sp.
Amaryllis sp.

Geographical Distribution. The first record of the occurrence of this virus was in Australia in 1915. Since that time it has been recorded from almost every part of the world, and it is stated to have been recognised for the first time in New York State so recently as the summer of 1935. The distribution is as follows : Australia—Victoria, New South Wales, South Australia, West Australia, Queensland and Tasmania ; British Isles and France, United States of America—Oregon, Wisconsin and California, and Canada—Ontario and Saskatchewan (12).

Control. The control of the diseases caused by *Lycopersicum Virus 3* may be approached from two aspects, according to whether the crops attacked are grown in the glasshouse or out-of-doors. In the former case the recommendations are briefly as follows : since it is upon the tomato plant that this virus is so frequently first introduced on to a grower's premises, new consignments of young tomato plants should be carefully inspected for signs of the disease. Any plants showing suspicious symptoms should be removed and burned. Whenever possible tomatoes should be grown in a house by themselves and not in the vicinity of miscellaneous ornamental plants. It is a very unwise procedure to move a crop of chrysanthemums or arum lilies into a glasshouse which has recently held tomatoes unless the house has first been thoroughly cleaned out and fumigated to kill the thrips vector. The thrips can be kept down under glasshouse conditions by fumigation with a good nicotine insecticide, or, alternatively, by the use of a dust containing nicotine or naphthalene. Either of

these is safer to use than an oil emulsion spray, which is liable to injure flowers and foliage.

The problem of the control of this virus out-of-doors is very much more difficult, since the insect vector cannot yet be adequately dealt with by means of sprays or dusts as it can under glasshouse conditions. It is possible that studies on the ecology of the thrips may yield data which would allow of intensive treatment applied over a shorter time. In California certain localities apparently function as endemic centres or foci of infection from which there may be considerable spread by thrips of the virus in the spring and summer. Such localities are characterised by mild winters, no summer rainfall, and the presence of living host plants throughout the year. Apparently the thrips are active in these centres throughout the year, but the virus is least abundant just after the winter rains, possibly because of a reduction in the thrips population. Besides certain ornamental plants and winter crops, some common winter weeds, such as mallow and chickweed, may harbour the virus. There is no indication that the virus is harboured in the native vegetation of uncultivated lands as is *Beta Virus 1* (curly top virus) (26).

At the moment the possibility of producing a variety of tomato resistant to virus or insect seems remote, though there is some evidence that the red-current tomato (*Lycopersicum pimpinellifolium*) shows resistance to infection (74).

Huissen Disease

The Huissen disease of tomatoes, so called from the place where it was first seen, is probably a composite virus disease of which *Lycopersicum Virus 3* (spotted wilt virus) is a likely component. The disease is characterised by sharply delimited, bronze coloured areas following the leaf veins and occasionally assuming the shape of rings, asymmetrical development of the leaves with curling of the margins, production of lateral shoots to replace the necrotic tops, premature death of the buds and flowers and a few circular lesions on the fruits, less distinctly defined than those of the spotted wilt virus (75).

LYCOPERSICUM VIRUS 4. Smith

Synonym. *Tomato Bushy Stunt Virus* (1936).

The Virus

This virus was described for the first time in 1935 (81, 88).



FIG. 54. *Lycopersicum Virus 4* (bushy stunt virus).

Tomato plant showing the bushy habit of growth characteristic of the later stages of the disease.

Resistance to Alcohol. The virus is inactivated by exposure for twenty-four hours to 90 per cent alcohol.

Serological Reactions. There is no serological relationship between this virus, *Nicotiana Virus 1* and *Solanum Virus 1* (Potato Virus X). The serological reactions of the virus have not yet been studied.

Thermal Death-point. Exposure for ten minutes to a temperature of 80° C., wet heat, inactivates the virus.

Resistance to Ageing. Infectivity is lost fairly rapidly upon storage in extracted sap at room temperatures. The virus concentrations as measured by local lesion counts show a steady fall during the ageing period. For example, a virus-sap suspension which gave numerous local lesions on the first day gave two lesions only on the twenty-fifth day and no infection at all on the thirty-third day. The virus retains its infectivity for long periods in extracted sap at low temperatures and shows no apparent reduction in concentration after storage for twenty-eight days at + 1° C.

Desiccation. The virus is inactivated when dried over sulphuric acid in a desiccator.

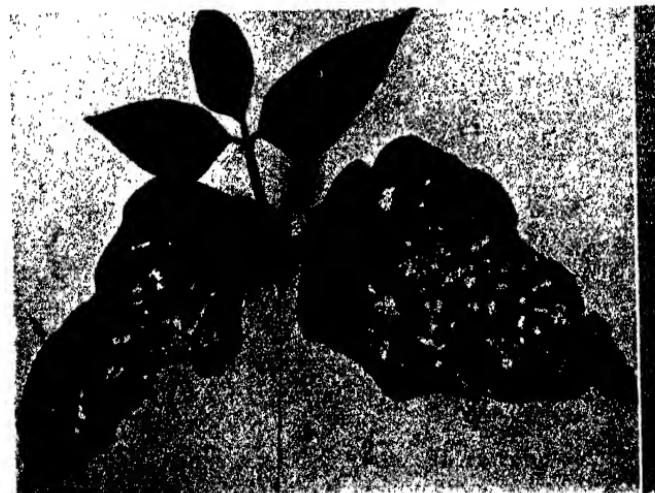
Particle Size. The virus is easily filterable and appears to be one of the smallest plant viruses known. The filtration end-point as measured on Gradocol membranes is 0·040 μ , which gives an average particle diameter of 14 to 20 m μ .

Transmission. The virus is sap-transmissible, but the insect vector has not yet been identified. The aphid *Myzus persicae* appears unable to transmit the virus. There is no evidence that the infective agent is carried in the seed.

Differential Hosts. The reaction of the black-seeded cowpea (*Vigna sinensis*) to this virus is very characteristic and serves to distinguish it from other viruses commonly infecting the tomato plant. Small lesions develop on the inoculated leaves three to four days after inoculation. These lesions at first pale, rapidly turn red at the edges and increase in size while still retaining their deep red edge and pale centre (see Fig. 55, B). Systemic spread of the virus in the cowpea has not been observed and symptoms appear to be confined to the inoculated leaves.

The symptoms induced by the virus on *Datura Stramonium* are quite characteristic, and in consequence this species is another valuable differential host. Circular or dendritic yellow spots appear locally about five days after inoculation and systemic invasion develops normally. The disease is a very severe one,

318 DISEASES CAUSED BY *LYCOPERSICUM VIRUS 4*



and the main characteristics are the crinkling and blistering of the leaves and the very marked ochre-yellow and green mottle. Affected *Datura* plants exhibit a very bold yellow and green variegation, and the normal shape of the leaves is lost (see Fig. 55, A). Occasionally a stem lesion develops, causing the plant to bend sharply to one side.

Diseases caused by *Lycopersicum* Virus 4

Solanaceæ

Lycopersicum esculentum. Tomato. *Bushy stunt disease*. Local symptoms develop on the inoculated leaf about five days after inoculation at a mean daily temperature of 60° to 70° F. These local symptoms usually consist of lesions either in the form of rings or circular necrotic patches. A little later the inoculated leaves become pale yellow, on which patches of green remain outstanding; frequently these leaves drop off. The development of the disease from this point seems to depend somewhat on the age of the tomato plant. In small plants, 3 to 4 inches high, there develops a general necrosis of the leaves together with yellow and purple coloration of the lower leaves and yellow spots on the young leaves. The lower leaves then wilt and the plant frequently dies. In young plants with soft sappy stems a gross necrotic lesion may develop at or about soil level, causing the plant to fall over (see Fig. 56, A). It is possible, however, that this lesion may be due to secondary causes. In older tomato plants the progress of the disease is less rapid and the stem lesion rarely develops. After the development of local lesions on the leaves there is an almost complete cessation of growth in height, the youngest leaves frequently become pale yellow in colour and twist over, sometimes being completely reversed. Occasionally a necrosis develops which kills the growing points; this is followed by growth of secondary shoots which produces the bushy or rosetted plant from which the disease gets its name of "bushy stunt" (see Fig. 54). The lower leaves next become chlorotic and

FIG. 55. *Lycopersicum* Virus 4 (tomato bushy stunt virus).

- A. Infected plant of *Datura Stramonium*, showing the bright mottling caused by this virus.
- B. Local lesions produced on the first leaves of cowpea (*Vigna sinensis*). The plants shown in A and B are valuable differential hosts.
- C. Ripe fruits of infected tomato plant.

usually undergo a colour change to yellow or purple which is highly characteristic. A common symptom is the appearance of concentric rings of bright yellow or purple, or, alternatively, necrotic lines of a purple colour develop along the veins on the yellow background of the leaf. The lower leaves finally shrivel or become completely chlorotic and drop off. The fruit does not always show symptoms, but when these are present they consist of a characteristic mottling or blotching of pale spots or ring-like marks on a darker background (see Fig. 55, C).

The outstanding characteristics of this disease in young tomatoes are cessation of upward growth and the yellowing and purplish coloration of the leaves. In older plants the same symptoms are present with the addition of lateral growth resulting in bushy and rosetted plants.

Nicotiana tabacum. Tobacco, var. White Burley. Local lesions develop on the inoculated leaves three days after inoculation at a mean daily temperature of 60° to 70° F. When they first develop, the lesions are small, red in colour and are surrounded by a yellowish halo ; they then rapidly dry out and become paper-white in colour. The lesions do not increase much in size as is the case with the lesions formed on *N. glutinosa* and cowpea. As a rule no further infection ensues and the plant remains healthy. Occasionally, however, in about 10 per cent of the inoculated plants there is a slight further development of the disease. A few scattered necrotic lesions may develop on the uninoculated leaves about fourteen days after the appearance of the local symptoms. Experiment showed that these lesions contain the virus, but that there was no virus in the intervening tissues. There is no mosaic mottling in affected tobacco plants and no systemic invasion in the usual sense of that term.

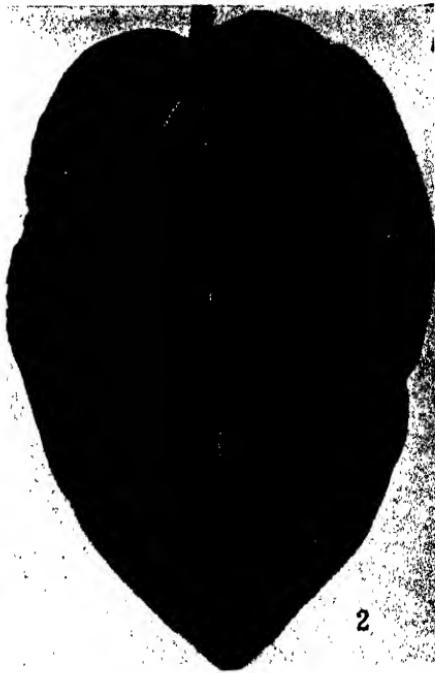
N. glutinosa. On this species small round lesions develop on the inoculated leaves forty-eight to seventy-two hours after inoculation. The lesions gradually increase in size, becoming quite large, sometimes measuring 5 mm. in diameter : at this stage

FIG. 56.

- A. Young tomato plant infected with *Lycopersicum Virus* 4 (tomato bushy stunt virus) ; note necrosis and collapse of lower leaves.
- B. Local lesions produced on a leaf of *N. glutinosa* by the same virus.
- C. Systemic infection of *Lycopersicum Virus* 8 (tomato spotted wilt virus) on tobacco, var. White Burley.



2



B



C

the lesions have a dark red edge and a pale centre (see Fig. 56, B). As in the case of similarly affected tobacco plants, there is usually no further development of the disease ; occasionally, however, a few scattered necrotic lesions develop on the younger uninoculated leaves. The virus is present in these lesions, but not in the intervening tissue.

N. langsdorffii. The behaviour of the virus on this plant resembles that in tobacco and the local lesions are similar. The development of systemic lesions has not been observed on *N. langsdorffii*.

N. rustica. This species appears to be resistant to infection with *Lycopersicum Virus 4*.

Miscellaneous Host Plants. No systematic study of the host range of this virus outside the Solanaceæ has been made, but experimental infections have been obtained on mimulus (Scrophulariaceæ) and on aster and zinnia (Compositæ). In all these plants only local lesions resulted and there appeared to be no further development of the disease.

Geographical Distribution. The virus has so far been recorded only from the British Isles, where it seems to be fairly widely distributed. Most of the records have come from commercial nurseries in the west of England and one from Northern Ireland. The writer has isolated the virus from *Datura* plants growing in a field near Cambridge ; this seems to suggest that the virus is insect-borne and that it is harboured by some wild host plant in the hedgerows from which it may have come in the first place to tomatoes.

Control. There is little to be recommended at present for the control of this disease other than the hygienic measures of eradication and destruction of infected tomato plants. Since the virus is quite infectious it must be remembered that the pruning knife or the hands of the workers are liable to spread the disease from affected to healthy plants. It is important in the case of a new virus disease such as this one that growers should be on the watch for it so that prompt eradication measures may be taken to prevent the virus assuming the epidemic character of *Lycopersicum Virus 1* (tomato streak).

LYCOPERSICUM VIRUS 5. Samuel, Bald and Eardley

Synonyms. *Tomato "Big Bud" Virus*, Samuel, Bald and Eardley, 1938 ; *Tomato Fruit Woodiness Virus*, Rischkow *et al.*,



FIG. 57. *Lycopersicum Virus 5* (tomato big bud virus).

Late stage of the big bud disease on a tomato plant from the field, var.
Marglobe.

(After Samuel, Bald and Eardley.)

1938; *Tomato Stolbur Virus*, Michailowa, 1935; *Tomato Virus 3*, J. Johnson's classification.

The Virus and its Transmission. The virus is not sap-transmissible, but is communicable by grafting or budding; no insect vector is at present known. There is no information available on the properties of the virus.

Diseases caused by *Lycopersicum* Virus 5

Solanaceæ

Lycopersicum esculentum. Tomato. *Big bud*. Tomato plants affected with this virus exhibit some remarkable teratological changes, particularly in the flowering parts. The first indication of infection appears at the tips of actively growing shoots. The youngest fruit truss, instead of becoming recurved as in normal plants, assumes an upright position. The buds on the truss also point in a vertical direction, the calyx segments remain united almost to the tips, and the whole calyx enlarges to a bladder-like form with a toothed opening at the top. A purple colouring due to the development of anthocyanin is often particularly marked along the veins of the bladder-like calyces, on the under sides of the young leaves and on the youngest portion of the stem. After a short time the axillary buds grow out, forming shoots affected in the same way as the main shoot. Simultaneously there is a gradual thickening of the stems of the affected parts due to the formation of an abnormal tissue in close association with the internal phloem (see Fig. 57).

The first effect of the virus on young developing leaves may be to stimulate the formation of more small lateral leaflets from the main and secondary petioles. There may also be some upward rolling of the leaf margins. After a time the youngest leaves become yellowish-green in colour, particularly at the margins, the lateral leaflets are often much reduced, and there is usually a purple colour underneath due to the formation of anthocyanin in the cells of the lower epidermis. As the disease progresses, smaller and smaller leaves are formed, until in the dense rosettes that are finally produced the leaves may be only 1 or 2 cm. long. Flowers that are fully formed at the time the virus invades the truss become erected from their recurved position, but undergo little change beyond a slight virescence of the petals appearing first along the mid-ribs. In younger flowers, just opening at this time, the corolla may become either stunted or enlarged and completely virescent. In both cases there is a cessation of further

development of anthers and ovary. Fruit already set at the time of invasion by the virus is immediately arrested in growth. Fruit already well developed but still green at the time of infection becomes hard and tough (*Tomato Fruit Woodiness* of Rischkow) and colours extremely slowly. In the younger fruit there may be a necrosis in the centres of the young embryos which are embedded in a firm parenchymatous tissue with thick cellulose walls instead of in a tissue which soon becomes gelatinous as in normal fruit (73).

Histopathology. There is a characteristic abnormal tissue which develops in association with the internal phloem. When a diseased stem is cut across this tissue can be seen as a band about 1 to 2 mm. across, internal to the wood, having a somewhat greenish, water-soaked appearance, owing to the close packing of the cells of which it is composed. The vegetative parts of the diseased tomato, with the exception of the leaf, are filled with starch grains. In "woody" fruits the structure of the mesophyll of the leaves and petals shows almost no intercellular passages. The thickness of the peduncle and the pedicel of diseased tomato plants exceeds that of a healthy plant, although the fruits are smaller. In the diseased fruits the fibro-vascular bundles are strongly lignified and the spiral vessels are replaced by porous vessels. The pollen from infected plants is sterile (64).

Solanum nigrum. Black nightshade. Samuel *et al.* report that *S. nigrum* has been found in the vicinity of infected tomatoes showing signs of the disease. Besides reduction in the size of the leaves and the presence of numerous axillary shoots, the flowering trusses were replaced by short branches with terminal papillæ of the same type found in affected tomatoes. An adventitious internal phloem tissue similar to that found in tomato "big bud" was also present.

Convolvulaceæ

Convolvulus arvensis. Bindweed. Bindweed plants have been observed by Michailowa (64) to be affected with symptoms suggestive of infection with *Lycopersicum Virus 5*. The diseased plants bear small round leaves which are chlorotic and often curl upwards. The histological changes in such plants are similar to those in diseased tomato plants, *i.e.*, reduced intercellular spaces, increased development of xylem and numerous starch grains in the tissue.

Geographical Distribution. The virus is present in all the Australian States, but is most commonly met with in New South

Wales and Victoria. An apparently similar disorder has also been recorded from Russia under the name of "Stolbur" disease.

LYCOPERSICUM VIRUS 6. McClean

Synonyms. *Tomato Bunchy Top Virus*, McClean; *Tomato Virus 2*, J. Johnson's classification.

The Virus

Resistance to Alcohol. Exposure of the virus in expressed sap for one hour to 30 per cent alcohol did not result in any appreciable loss of infective power. Alcohol in excess of 30 per cent appears to cause some destruction of the virus.

Thermal Death-point. The virus is inactivated by ten minutes' exposure to temperatures above 70° C. Some destruction of the virus occurs at temperatures between 60° and 70° C., but temperatures below 60° C. have little effect.

Resistance to Ageing. Inactivation of the virus commences after the expressed sap has stood at laboratory temperatures for twelve to twenty-four hours. Up to twelve hours' ageing the infectivity of the virus remains high.

Filtration. The virus does not easily pass a Chamberland L₁ or Seitz filter.

Transmission. This virus is easily transmitted by sap inoculation, but the insect vector has not yet been definitely identified. There is no evidence that the virus is carried in the seed.

Differential Host

Nicotiana glutinosa is susceptible to infection and the symptoms on the leaves are not pronounced, but the flower colour shows a characteristic "break" of pink or white stripes on a yellowish background (see Fig. 58, C).

Diseases caused by *Lycopersicum* Virus 6

Solanaceæ

Lycopersicum esculentum. Tomato. *Bunchy top*. The first indication of the disease is a sudden and almost total cessation of growth at the branch extremities, with the result that at these points the leaves become closely crowded, giving the plant the bunched appearance typical of the early stages of infection. The accompanying modifications of the leaves are characteristic. Those fully developed at the time of infection do not undergo any change,



FIG. 58. *Lycopersicum Virus 6* (tomato bunchy top virus).

- A. Affected plants showing a large number of laterals bearing small leaves.
- B. Lower surface of a leaflet showing blackening of main and lateral veins.
- C. Flower from infected plant of *N. glutinosa* showing "breaking" of the flower colour.
(After McClean.)

but remain normal in appearance. Thereafter in passing up through the condensed region, there is a progressive decrease in their size, and closer crowding of the leaflets on the rhachis. The leaflet margins become curled towards the under surface, the tips frequently are twisted downwards and the surfaces show a puckered condition. The axillary buds, particularly those of the lower leaves, are forced into early activity. The check on the upward growth of the axis is not permanent and is followed by a definite elongation of the internodes with the production of a somewhat spindling type of growth. A plant at this stage of the disease frequently shows the following differentiation : (1) A lower region apparently normal, with the exception of axillary shoots, and bearing normal-looking leaves ; (2) a middle region characterised by the condensation of the axis, bunching of the foliage, a progressive reduction in size of the leaves, and various forms of leaf distortion ; (3) an upper region in which the internodes have again lengthened, characterised by a thin axis and small leaves which show little or no distortion. Axillary shoots which develop subsequent to infection, whether from the lower normal leaves or from those visibly affected, will be modified in a manner similar to the main axis in the upper region (see Fig. 58, A).

Thus if the tomato plant is infected early, then in its later stages it will be composed of a varying number of thin upright axillary branches, while one infected late will have a weak habit and its thick semi-procumbent stems will be clothed with numbers of short axillary shoots bearing the typical small leaves. Necrosis of leaves and stems also appears to be a characteristic of the disease, especially in the early stages, and develops with greatest severity on those leaves showing the first signs of a reduction in size. Both main and lateral veins may be affected and the necrosis frequently spreads laterally into the mesophyll forming irregular black areas (see Fig. 58, B).

The flowers of affected plants do not appear abnormal in any way, but the fruits are small, occasionally distorted and of no commercial value. Such fruits are either seedless or else contain a few small seeds, only a proportion of which may be fertile. The outstanding characters of this disease are the dwarfing, extreme reduction in leaf size, streak necrosis and various forms of leaflet distortion (61, 62).

Physalis viscosa L. Infected plants show a definite lag in growth and no increase in the number of shoots, and their initial shoots tend to produce a number of short lateral branches which

flower prematurely. Four months after infection the shoots of infected plants are stunted and much-branched and bear small leaves. The fruits are fairly normal in appearance, but are smaller than those of healthy plants. The reaction of the plant in general to the virus is more severe under open-air conditions.

P. peruviana L. Cape gooseberry. There appear to be two types of reaction in this species to the virus. In one type the virus seems to have little or no effect on the plant except for a slight retarding influence on growth. In the other type of reaction a severe disease is produced characterised by severe stunting, reduction in leaf size and bunching of the foliage. This is followed, later, by the development of numerous short axillary shoots and the dying of the older normal leaves. No fruits are formed. McClean makes two suggestions to account for the severe disease; one is that it is caused by a modified form, possibly another strain, of the virus, and the other is the possibility of contamination with a second virus, although this is considered under the circumstances to be improbable.

Nicandra physaloides Gaertn. Diseased plants remain small, produce small leaves and a number of short lateral branches. The production of flowers and fruit is not affected.

Nicotiana tabacum. Tobacco. The tobacco plant, several varieties of which were tested by McClean, is susceptible to infection by the virus, but appears to carry it without visible symptoms. On transmission of the virus back to tomato from tobacco the virus seemed to have acquired an added virulence.

N. glutinosa. See under "Differential host."

Other Solanaceous Host Plants. The following cultivated species of the Solanaceæ are liable to infection, and all carry the virus without symptoms: *Petunia hybrida*, *Solanum melongena* (egg plant), *S. tuberosum* (potato) and *Capsicum annuum*. Various wild species of this family are susceptible, and these also show little effect of the infection other than a tendency to the production of smaller leaves and slower growth than normal. The following wild Solanaceous plants have been experimentally infected with the virus: *Solanum aculeastrum* Dunal., *S. aculeatissimum* Jacq., *S. incanum* L., *S. nigrum* L., and *S. sodomæum* L.

Compositæ

Zinnia elegans Jacq. The zinnia appears to be susceptible to infection with this virus, but the plants remain normal in appearance.

Geographical Distribution. The virus has so far been recorded only from South Africa, where it has been found more or less generally distributed through the low-lying districts of the eastern part of the Transvaal. There are no records of its occurrence outside this area, either in the same province or other parts of South Africa.

Control. The only method of control available consists in paying strict attention to the ordinary hygienic cultivation necessary when dealing with this type of infectious disease. The avoidance of successive handling of diseased and healthy plants is important, and all infected plants should be carefully rogued out and destroyed. In South Africa where this disease is prevalent the tomato is an outdoor crop, and therefore it is advisable to eradicate Solanaceous weeds in the vicinity, since they may act as the source of an initial infection.

The Composite Virus Diseases of the Tomato

Two composite virus diseases of the tomato only are dealt with here.

Tomato Streak Caused by Nicotiana Virus 1 and Solanum Virus 1. The tomato streak disease caused by the interaction of these two viruses is symptomatically identical with the disease caused by *Lycopersicum Virus 1* (tomato stripe virus). Gross lesions develop longitudinally in the stem and necrotic spots appear on the leaves. Young plants are usually killed by this disease. Tomato streak is not of common occurrence in England, where opportunity for infection with both viruses is small, but the disease is of serious importance in America.

Disease due to the Combined Action of Nicotiana Virus 1 and Cucumis Virus 1. Affected tomato plants are abnormally short and compact, the leaves just below the growing point being characterised by an upright, lusty habit, due in part to very short internodes and partly to an excessively twisted and erect development of the petioles. The young leaflets are curled, distorted, thick, with yellow veins, and occasionally show the filiform malformation associated with *Cucumis Virus 1*, while the older leaves are sharply rolled upwards at the margins and exhibit a mild yellowish-green associated with ordinary tomato mosaic (*Nicotiana Virus 1*). The leaves may show a peculiar greenish-purple coloration, especially along the veins and on the underside, while other leaves show large yellow bleached-looking spots and a russet coloured necrosis bordering the larger veins. The flowers

are commonly malformed and abortive, but some fruit may be produced which is deeply ridged and when small shows a characteristic pointed protuberance at the blossom end (20).

HYOSCYAMUS VIRUS 1. Hamilton

Synonym. *Hy. III Virus*, Hamilton, 1932.

The Virus

Thermal Death-point. The virus survives exposure of ten minutes to a temperature of 50° C., but not to a temperature of 60° C. or over.

Resistance to Ageing. Infectivity is retained for a comparatively short time in clarified sap, not more than twenty-four hours.

Filterability. The virus will pass an L₁ Pasteur-Chamberland filter candle, but not an L₃.

Transmission. The virus is sap-inoculable and the insect vector is the aphid *Myzus persicae* Sulz. (see p. 538). There is a delay in development of infective power within the insect which seems to vary from three days in the autumn to thirty days in the spring. In some recent studies on the relationship between the aphid and this virus, Watson (107) gives the following facts : A maximum percentage infection was obtained during the winter months and a minimum during the summer months. The percentage infection increases with the number of aphides used per plant, and the relation between the number of infections obtained for each aphide-number indicates that the infections are local and independent. The percentage infection increases with increased feeding time on the healthy plant, but there is no indication of a preliminary time period in which no infection is obtained. The percentage infection decreases very rapidly with increasing time on the infected plant from two minutes to one hour. After one hour it increases slightly with further increase of the feeding periods. *Myzus persicae* is capable of infecting two consecutive plants without intermediate access to a source of infection, but the number of second infections decreases rapidly with increasing time on the healthy plant, and is negligible after one hour.

Diseases caused by Hyoscyamus Virus 1

Solanaceæ

Hyoscyamus niger. Henbane. Primary symptoms take the form of clearing or yellowing of the veins of the youngest leaves ;

this is followed by a yellow mosaic and dark green banding of the veins.

Nicotiana tabacum. Tobacco. The primary symptoms in tobacco also appear as clearing of the veins, and this is followed by a yellow mosaic with broad, blistered, dark green bands. As the plant grows older, the bands become smaller and often necrotic at the edges, and the leaf exhibits a confused chequered design of dark bands, yellow mottle and necrotic spots. In old tobacco plants the young leaves may frequently carry the virus without symptoms. Intracellular inclusions, or X-bodies, are usually present.

Nicotiana glutinosa. This plant reacts with a systemic infection of vein-banding symptoms which tend to become faint in older leaves. A characteristic of the disease in this species is the "breaking" of the flower colour, which, though normally self-coloured pink, becomes white streaked with pink. The symptoms take longer to develop in *N. glutinosa* than in the other species, fourteen or fifteen days as compared with five or seven days.

Nicotiana glauca. The virus causes a violent necrosis in this plant, accompanied by blistering of the leaves and stunting.

Petunia sp. A yellowing of the regions round the veins is produced in petunia, together with necrosis in the older leaves.

Datura Stramonium. This plant reacts similarly to *Hyoscyamus* with a yellow mosaic, a tendency to dark green banding of the veins and blistering.

Geographical Distribution. This virus has only been recorded from Harpenden, England, where it was isolated from a field of *Hyoscyamus* which was being grown for commercial purposes (30).

DATURA VIRUS 1. Smith and d'Oliveira

The Virus

So far as the writer is aware, this virus has not been described before, and the following account is from some unpublished work by Smith and d'Oliveira. The virus was first isolated in 1936 from some *Datura* plants which were being grown for experimental purposes in a field near Cambridge.

Thermal Death-point. Exposure of extracted sap for ten minutes in a water-bath at a temperature of 50° C. does not inactivate the virus, but infection is destroyed by similar exposure to a temperature of 55° C.

Dilution End-point. The virus is not inactivated at dilutions of 1 : 5,000, but loses infectivity at 1 : 10,000.

Resistance to Ageing. The virus remains infective for nine days, but not for twelve days, in expressed sap at room temperatures.

Transmission. The virus is sap-transmissible and is fairly easily transferred from plant to plant without the aid of an abrasive. Whenever the infective sap is taken from plants of *Nicotiana*, tomato or *Phaseolus*, symptoms develop in all inoculated susceptible plants, except *Datura*, in five to six days. On *Datura* symptoms always take twelve to fifteen days to develop under similar conditions; when sap from an infected *Datura* is used for inoculation, symptoms on this and all other susceptible plants are visible only after a period of twelve to fifteen days.

There is a certain amount of evidence which suggests that the insect vector is the aphis *Myzus persicae* Sulz., but this is not yet proved.

Differential Host

Phaseolus vulgaris. French or snap bean. Small roundish lesions develop on the inoculated primary leaves of the bean. These lesions do not increase much in size and do not spread along the veins in the manner of *Nicotiana Virus 11*. If the infection is heavy, a severe systemic necrosis develops and the leaf rapidly shrivels and dies. This is followed by the necrosis of the vascular system in the stem and the whole plant is killed.

Diseases caused by Datura Virus 1

Solanaceæ

Datura Stramonium. Thorn apple; Jimson weed. A slight yellow flecking develops on the inoculated leaves of *Datura* after twelve days. In the next three or four days the lesions become strongly necrotic and quite large areas of the leaf blade may be killed. The leaves, however, remain attached to the plant. As the lesions slowly increase in size, they may coalesce, but there is no spreading along the veins. Old lesions are entirely dried out and have a white parchment-like aspect. Systemic infection, which does not always develop, consists of a yellow flecking which does not become so necrotic as the primary symptoms (see Fig. 59, A and B).

Nicotiana tabacum. Tobacco, var. White Burley. The formation of local lesions on the inoculated leaves is not a constant feature, but when present they assume the form of small, necrotic specks of rather irregular shape. A clearing of the veins is a frequent first sign of infection. The leaves which show this vein-

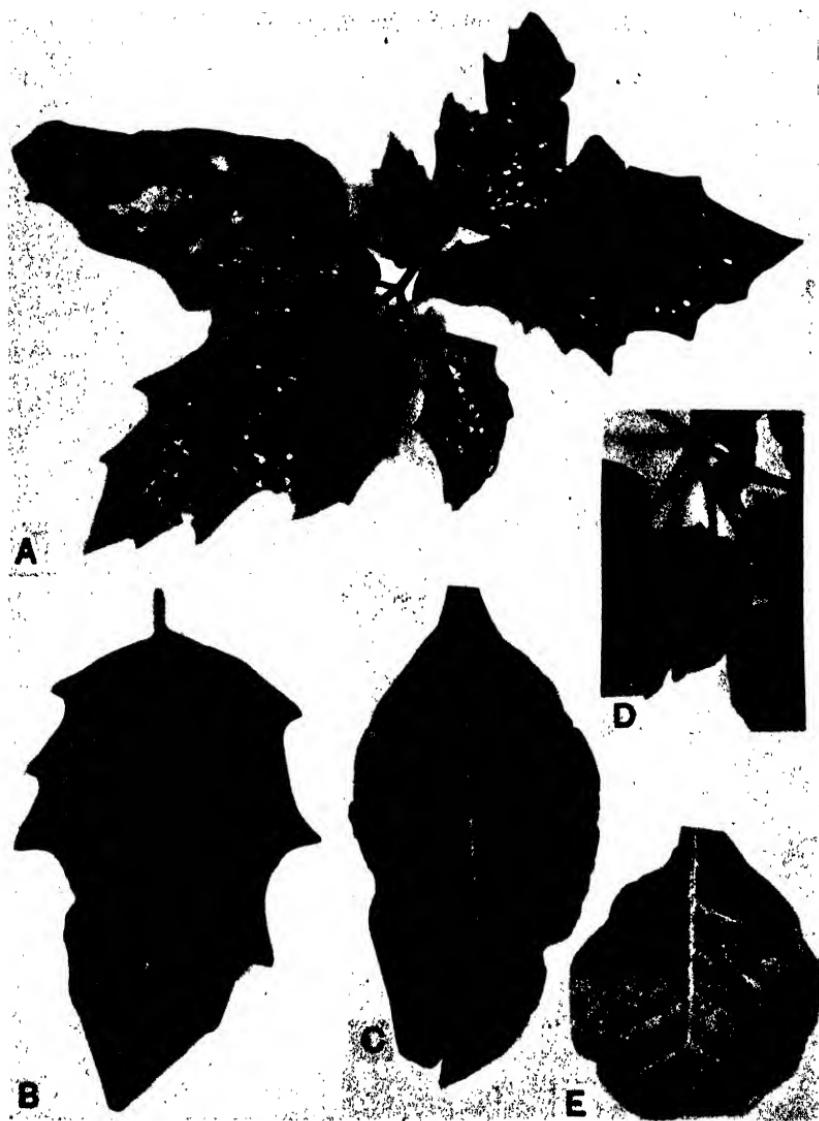


FIG. 59.

A and B. Systemic infection of *Datura Virus 1* on *Datura*.
C. Systemic infection of the same virus on White Burley tobacco.
D and E. Systemic infection of *Nicotiana Virus 15* on *N. glutinosa* and White Burley tobacco respectively : note the characteristic necrotic etching in the latter.

clearing become covered with numerous necrotic spots which do not increase much in size, but become scattered as the leaf blade enlarges (see Fig. 59, C). The general growth of the plant is not much affected and later-formed leaves are usually symptomless; such leaves, however, contain the virus in high concentration. Sudden outbreaks of symptoms may appear on these apparently recovered plants, in such cases an oak-leaf pattern may be formed, the border line of which is dotted with small necrotic specks. Alternatively the border line may itself form single rings measuring about 1 cm. in diameter. In some of its phases the disease resembles that produced in tobacco by *Solanum Virus 1* (potato virus X), but tests have shown that there is no cross-immunity between the two viruses.

N. glutinosa. When local lesions are formed on this plant they appear as chlorotic rings or half-rings which may later become slightly necrotic. Systemic infection produces a crinkle, together with moderate mottling; affected leaves are undersized. Infected plants lose their symptoms after a time, but remain stunted. No fresh outbreak of symptoms in such plants has been observed.

Lycopersicum esculentum. Tomato. Small necrotic lesions develop on the inoculated leaves; these lesions do not increase in size, but remain perfectly round and of a brownish colour. In older plants no systemic spread of the virus has been observed. When very young plants are inoculated, however, the virus does become systemic, producing minute brown specks on the newest leaves. From there the virus spreads and enters the growing points, which are killed out.

Solanum tuberosum. Potato. It has not been found possible to infect the potato plant with this virus, either by sap-inoculation or by grafting.

Leguminosæ

Vigna sinensis. Black-seeded cowpea. Local lesions are produced on this plant, similar in appearance to those formed on *Phaseolus vulgaris*. Systemic symptoms are much less severe.

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CHAPTER VI

THE VIRUSES AFFECTING THE POTATO PLANT

Solanum Viruses 1-18 ; The Composite Mosaic Diseases of the Potato.

UNDER the system of classification adopted in this book, *Solanum Virus 1* is the first of that very difficult group of viruses which attack the potato plant. Chiefly owing to the extremely variable response of the many potato varieties to infection with the different viruses and to the fact that potato plants are so often infected with a complex of viruses, one or other of which is frequently "carried" without the exhibition of symptoms, the knowledge of this group of diseases is still in a state of flux. This account, therefore, is offered as being the best that the writer can compile from existing knowledge, and is not intended to be a complete or final statement.

It is apposite to make here a reference to the valuable early exploratory work of Orton (37, 38), who first described potato mosaic and streak, and who, with Quanjer and Appel, was the father of modern potato virus work.

Since this group of viruses is largely confined to the potato, attention will be mainly devoted to the diseases caused by them in that plant; the differences in varietal reaction to the same virus are carefully described.

In a separate section the composite virus diseases of the potato such as crinkle, rugose mosaic, etc., are dealt with, the symptoms are described and the viruses concerned in the complex are detailed.

SOLANUM VIRUS 1. Orton

Synonyms. *Potato Virus X*, Smith, 1931 ; *Simple Mosaic Virus*, Murphy, 1932 ; *Potato Acronecrosis Virus*, Quanjer, 1931 ; *Healthy Potato Virus*, Valleau and E. M. Johnson, 1930 ; *Latent Potato Virus*, Schultz, 1925 ; *Tobacco Ringspot Virus*, J. Johnson ; *Interveinal Mosaic Virus*, Quanjer ; *Potato Virus 16*, J. Johnson's classification.

The Virus

Solanum Virus 1 is the most ubiquitous of all the potato mosaic viruses and forms part of the basis of most of the severe potato mosaic diseases. It occurs apparently throughout the world, and in some countries is so widespread that no plant of established commercial potato varieties has been found free of it. In the case of new varieties originating from the true seed, it is easy to find plants free from this virus, and the presumption is that most, if not all, varieties at least start free from infection. Subsequently they contract it at a greater or less rate, depending on susceptibility, climate, and probably chance associations in their early days. The varieties Up-to-date and British Queen are about the same age, but it is comparatively easy to find plants of the latter variety still free of the virus, and this is probably due to some factor of resistance. Murphy (in litt.) states that he has observed the content of this virus in Arran Banner rise from *nil* to 95 per cent after seven years' growth in Donegal.

This virus, like many others, exists in a number of closely allied strains (24, 46, 55A). That these strains are, however, fundamentally the same virus is shown by their serological and immunological reactions. Their physical properties, also, are similar and the chief difference lies in the severity of the respective symptoms produced in the tobacco plant, which is a valuable differential host.

Resistance to Various Chemicals. *Alcohol.* The virus is inactivated after twenty-four hours' exposure to 85 per cent ethyl alcohol.

Nitrous acid causes loss of activity without affecting the reactions of the virus with antiserum (7A).

Enzymes. In purified suspensions the virus is completely inactivated by a 0·02 per cent solution of pepsin in three hours at pH 4 and 38° C. Incubation with crystalline preparations of trypsin also destroys infectivity. Both these enzymes destroy the power of the virus to react with antiserum. Papain alone and cyanide alone have no effect on the virus, but the two together inactivate it.

The inactivation by the enzymes occurs only at pH 8, at which these are proteolytically active, and the extent is directly proportional to the concentration of the enzyme and to the time of incubation (7).

Serological Reactions. Spooner and Bawden (57) have demonstrated the presence of a common antigen in the saps of tobacco,

Nicotiana glutinosa, *Datura Stramonium* and the potato varieties President and Up-to-date, infected with *Solanum Virus 1* (potato virus X). Anti-viral sera in a dilution of 1/10 neutralise the infectivity of purified virus suspensions; anti-healthy tobacco sera and normal rabbit serum do not. The virus antigen is specific to this virus and the closely related *Solanum Virus 6*. No antigenic differences were found between the different strains of this virus (6, 7A).

Thermal Death-point. The virus is destroyed by exposure for ten minutes at 66° C.

Dilution End-point. With extracts from young recently infected tobacco plants positive infections are seldom obtained at a greater dilution than 1 : 10,000.

Resistance to Ageing. The virus remains infective in sterile sap at room temperature for periods of two to three months.

Desiccation. The virus in the unpurified state is destroyed by drying.

Filterability and Particle Size. The virus is filterable through Pasteur-Chamberland candles L₁, L₃ and L₅. The particle size as measured by the Elford ultra-filtration process is 75 to 112 μ (56).

Transmission. *Solanum Virus 1* is sap-inoculable; there is no evidence of seed transmission. It can be transmitted by core grafts in the tuber. The exact method of the spread of the virus in the field is not yet known, and in view of its world-wide distribution this is a most curious fact. None of the common sap-sucking insect fauna of the potato plant is able to act as a vector. Out of about 100 transmission tests with various species of thrips, however, the writer has had, or appeared to have, five positive infections. It is possible that a species of thrips is actually the vector, but that infection is conveyed by the insect feeding in the flowers. It is interesting to find some circumstantial evidence in support of this theory in observations made by Dr. T. McIntosh, of Edinburgh. He has noticed that the following potato varieties, all of which are free-flowering, are most frequently naturally infected with the virus: Majestic, Dunbar Cavalier, Golden Wonder, Up-to-date and Kerr's Pink. On the other hand, certain varieties which seldom flower, at all events in Scotland, remain comparatively free of the virus, such are King Edward, Great Scot and Epicure.

Differential Hosts

Among potato varieties, Arran Crest, Epicure and King Edward

are very susceptible to artificial infection ; they develop a severe top necrosis and are usually killed. The disease, however, is never seen in these varieties in the field.

Among other Solanaceous plants, *Datura Stramonium*, tobacco, var. White Burley, and *Nicotiana glutinosa* are useful indicators. On all three species virulent strains of *Solanum Virus 1* produce the typical "ringspot" symptom frequently accompanied by a severe general necrosis. On tobacco the rings are especially clear. As already stated, however, this virus occurs in several strains of varying virulence, and some of the milder types produce only a fleeting mottle on these plants without ring formation or necrosis. On potatoes, however, the difference in severity of the symptoms produced by the various strains is not so great.

Diseases in Different Potato Varieties caused by *Solanum Virus 1*

This is the virus most frequently found in naturally infected potatoes, and few commercial stocks, other than those of the varieties Arran Crest, Epicure, Great Scot and King Edward, are free from it. It is almost universally present in the commercial potato stocks of America, where it is known as the "healthy potato virus." The majority of the varieties either carry the virus without symptoms or, when infected, show an interveinal mottle with but little dwarfing of the plant or deformation of the foliage (see Fig. 60, A).

King Edward. This variety develops acute necrosis of the growing points within fifteen days and usually dies. The tubers may be free of lesions or else show a destructive necrosis with cork formation. In the second year, tubers of infected plants either fail to sprout or else produce healthy plants. It must be remembered that King Edward varieties of potato appear to be invariably infected with *Solanum Virus 7* (paracrinkle virus), so that it cannot be said definitely whether the foregoing top necrosis disease is caused by *Solanum Virus 1* alone or the two acting in unison.

Arran Crest and Epicure. These varieties react similarly to the above and are usually killed. Plants in the second year of infection are acutely necrotic and soon die. This extreme susceptibility really means a practical immunity, since the plants are either healthy or killed outright.

Histopathology. Intracellular inclusions, or X-bodies, are frequently present in the mottled areas of potato plants infected with *Solanum Virus 1*, particularly in plants which have been

B



A

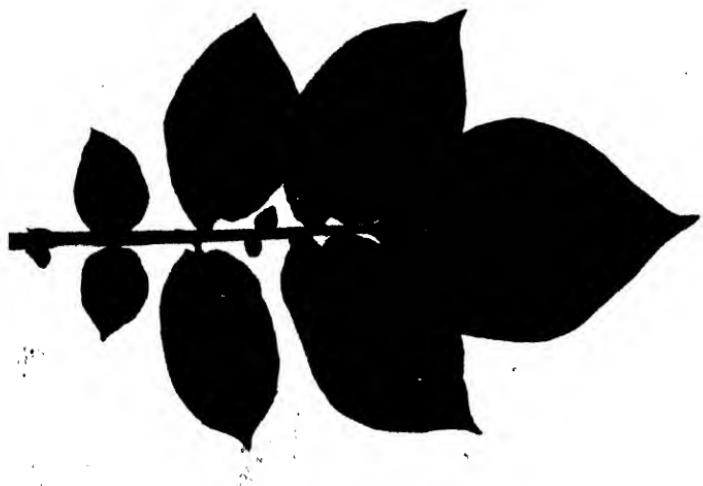


FIG. 60.

A. *Solanum Virus 1* (potato virus X).
B. *Solanum Virus 2* (potato virus Y). Both on potato President.
(A, after Murphy.)

diseased for some time. When definitely formed, the X-bodies appear as conspicuous masses in epidermal and hair cells of the leaf, in the palisade and spongy parenchyma and in the parenchymatous tissue of the veins. They have a homogeneous or finely granular structure and stain like cytoplasm. The typical shape is more or less rounded, but elongated and irregular shapes also occur. There is rarely more than one in a cell. They are situated close to the nucleus, from which, however, they are clearly distinguishable. Numerous vacuoles are present in the X-bodies, some large and some very small. The bodies vary in diameter from 6μ to about 50μ , the average size being about one and a half times to twice the size of the nucleus (12).

In the top necrosis disease an examination of the necroses in the stems shows that they arise in the phloem, usually, but not always, in the internal phloem, and from this they spread into other neighbouring tissues. The first pathological symptom is the thickening of the cell walls of the primary phloem elements. This thickening is accompanied by the separation of adjacent primary cell walls. Thus intercellular spaces are formed, and these are filled with a yellowish-brown gum-like deposit. The necrotic process passes into the neighbouring parenchyma, and it is in this, owing to the greater size of the cells, that the formation of intercellular spaces is most pronounced. The necrosis spreads from the internal phloem in all directions, but most markedly through the perimedullary zone towards the wood. The wood parenchyma is particularly susceptible, and the cells may collapse completely or become filled with a plastic material similar to that mentioned above. Necrotic changes also originate, though with less frequency, in the outer phloem, the main spread of the necrosis being also towards the wood (see Fig. 68, D).

In necrotic tubers from top necrosis plants the necroses develop in essentially the same manner as those in the stem, the first symptom being a yellowing and thickening of the walls of the phloem group. The necrosis spreads rapidly to the storage parenchyma, and the phloem bundles collapse. The contents of the cells disappear and the walls are suberised (8).

Diseases in Other Solanaceous Plants caused by *Solanum Virus 1*

Lycopersicum esculentum. Tomato. The severity of the symptoms produced in the tomato plant depends upon the virulence of the strain of virus infecting it.

The most virulent, or S strain, produces a necrotic disease in young tomato plants. Local necrotic lesions may develop on the inoculated leaves, and these are followed by a definite mosaic mottle with some necrosis and occasional necrotic concentric rings. The less virulent virus strains produce only a dark and light green mottle, which in the mildest cases is very faint and fleeting.

Petunia sp. Garden variety. Certain species of petunia are extremely resistant to infection with *Solanum Virus 1*, although they are probably not immune. This resistance is important in separating out virus mixtures.

Hyoscyamus niger. Henbane. This species is easily infected with the virus and is frequently killed by the more virulent strains. Large necrotic lesions may develop on the inoculated leaf, followed a few days later by ring-like lesions on the other leaves. The less virulent strains of the virus produce only a dark and light green mottle.

Datura Stramonium. This plant is very susceptible to infection and reacts to the virulent virus strain with great violence. The most severe reaction takes the form of gross lesions, while occasionally the whole leaf is killed by a severe necrosis of the "scorch type." The more usual reaction is the development of small double rings on the middle leaves; these rings rapidly disappear with continued growth of the plant, and their place is taken by a mosaic mottle of dark and light green or yellow, often accompanied by a darkening of the green colour, especially near the veins.

The milder strains of the virus produce correspondingly mild symptoms which appear as a faint light and dark green mottle without necrosis. *Datura* is a useful indicator plant for testing for the presence of this virus. Intracellular inclusions are frequently present.

Nicotiana tabacum. The reaction of the tobacco plant to this virus is very similar to that of *Datura*, varying with the virulence of the virus. The ringspot type of symptom is shown to a greater degree in the tobacco plant, but mild strains of the virus produce only a faint mottle very similar to the vein-banding symptoms caused in the same plant by *Solanum Virus 2* (potato virus Y).

Solanum dulcamara. Woody nightshade. Although this plant is easily infected by mechanical inoculation, the writer has never observed it to be naturally infected in the field. The

disease develops in about ten days under glasshouse conditions with dark circular lesions on the inoculated leaves. Later isolated concentric rings with a central spot may develop on the other leaves. As the plant grows, symptoms tend to disappear and the plant acts as a carrier of the virus.

S. nigrum. Black nightshade. The usual symptoms on *S. nigrum* consist of a faint mottle of dark and light green patches, occasionally there are a few necrotic rings.

SOLANUM VIRUS 2. Orton

Synonyms. *Potato Virus Y*, Smith, 1931; *Streak Virus*, Orton, 1920; *Leaf-drop Streak Virus*, Murphy, 1921; *Stipple-streak Virus*, Atanasoff, 1922; *Acropetal Necrosis Virus*, Quanjer, 1931; *Hy. II. Virus*, Hamilton, 1932; *Vein-banding Virus*, Valleau and Johnson, 1930.

The Virus

Resistance to Chemicals. The virus is inactivated by 75 per cent alcohol.

Serological Reactions. It has not yet been found possible in England to test the serological reactions of *Solanum Virus 2*. Chester (11), however, states that he has found the serological reactions of this virus to be similar to those of *Cucumis Virus 1* (cucumber mosaic virus), but since he used as a test plant *Datura Stramonium*, which is immune to the virus in question, it is probable that he had some other virus under consideration.

Thermal Death-point. The virus is inactivated after exposure to 52° C. for ten minutes.

Dilution End-point. Infectivity begins to fall off rapidly at dilutions of 1 : 100 and positive infections are only occasionally obtained at dilutions of 1 : 1,000.

Resistance to Ageing. The longevity *in vitro* of the virus in expressed sap at room temperature is only twenty-four to thirty-six hours. This rapid inactivation is probably partly due to oxidation effects, and it can be slowed down by the addition of reducing agents and by the effects of low temperature.

Desiccation. The virus is inactivated by drying.

Filterability. Owing to its high capacity for adsorption, the virus cannot be filtered through candles. It has been filtered with great difficulty through Gradocol membranes of 0.42 μ average pore diameter.

Transmission. The virus is sap-transmissible, and Reddick (42) considers that it may be carried to a small percentage in the true seed of the potato. The insect vector is the aphid *Myzus persicae* Sulz. (see p. 538). The relationship between this virus and the insect is the same as that existing in the case of *Hyoscyamus Virus 1* (see p. 331).

Differential Hosts

Nicotiana tabacum. On tobacco the first symptom is a clearing of the veins of the youngest leaves seven days after infection. This is followed in a day or two by a characteristic banding of the veins of the older leaves. There is no necrosis.

Solanum nodiflorum. This plant reacts to infection with *Solanum Virus 2* with a pronounced veinal mosaic.

Datura Stramonium is immune to infection with the virus, and this makes the plant a useful one for eliminating the virus, from a complex (see p. 394).

Diseases in Different Potato Varieties caused by *Solanum Virus 2*

In a large number of different potato varieties the symptoms produced by infection with this virus are of the leaf-drop streak type, or acropetal necrosis. This is one of the commonest virus diseases in English potato fields, and in severe epidemics affected fields appear as though attacked by "blight," *Phytophthora infestans*.

President. Symptoms commence to develop about three or four weeks after infection and take the form of a blotchy mottle spreading from the veins and affecting the topmost leaves only. Concurrently with this, or a little later, fine necroses appear along the veins on the undersides of the leaves (see Fig. 60, B). These necroses increase in severity and spread along the course of the veins; a little later they penetrate the leaf tissue and become evident on the upper surface. The necrosis then passes down the petiole and reaches the main stem, producing thereon brown longitudinal lesions. The leaves become completely necrotic and withered, but remain hanging as if attached to the stem by a thread. The topmost leaves are not necrotic, but are frequently mottled and slightly crinkled. This description applies to the current season infection; in subsequent years there may be little necrosis or leaf-drop, but infected plants are small and stunted and their leaves and stems are very brittle.* The internodes are

short, and the leaves are generally mottled, twisted and bunched together. The whole plant is dwarfed and rosetted. The tubers are not necrotic and appear normal.

Histopathology. The cell wall in the necrotic area becomes suberised and the cell contents either disappear or are changed into a gummy mass filling the cell lumen. The spread of the necrosis is mainly perpendicular and not, as in top necrosis, mainly towards the wood ; it thus gives rise to dark superficial stripes, visible under the epidermis, which may itself remain green. The necrosis further spreads around the stem along the collenchyma, forming narrow arc-like stripes, as seen in transverse section (see Fig. 68, C). No intracellular inclusions, or X-bodies, have been observed in the cells of plants affected with acropetal necrosis. In leaves of President plants showing this disease curious circular sac-like deposits of crystals are sometimes found. These are of considerable size and occur in the same leaf, both in the completely necrotic tissue and in that still otherwise normal (3).

The following potato varieties react to *Solanum Virus 2* in a similar manner to President : British Queen, Arran Banner, Majestic, Up-to-Date ; the American varieties, Irish Cobbler, White Rose, Burbank, Bliss Triumph, Green Mountain and Earliest of All (see Fig. 61).

Salaman (45) has grouped the reactions of *Solanum Virus 2* on a number of potato varieties as follows :

No reaction. Di Vernon, Kerr's Pink.

Veinal necroses only. International Kidney, Sharpe's Express.

Veinal necroses and leaf-drop streak. Arran Consul, Arran Crest.

Mosaic or crinkle, sometimes accompanied by small necrotic spots. Arran Victory, Duke of York, Epicure, King Edward.

Mosaic and veinal necroses. Abundance, Arran Chief, Great Scot.

Mosaic or crinkle, veinal necroses and leaf-drop streak. Arran Banner, British Queen, Up-to-Date, King George, Majestic, President.

Diseases in Other Solanaceous Plants caused by *Solanum Virus 2*

The diseases produced in tobacco and *S. nodiflorum* by this virus have already been described, so a further description is not given here.



FIG. 61. *Solanum Virus 2* (potato virus Y).
(Left) Potato Up-to-Date showing primary symptoms of infection ; note the brown lesions developing on the under surface of the leaves.
(Right) Potato Eclipse showing secondary or systemic infection ; note leaf-drop effect.

Solanum dulcamara. Woody nightshade. The virus cannot apparently be transmitted to this plant by sap-inoculation, but it can be transferred by graft. The virus is carried without symptoms, but its presence can be demonstrated by grafting from such a carrier back to the President potato.

S. nigrum. Black nightshade. This plant is susceptible to infection by sap-inoculation. The initial symptom of vein-clearing is hardly discernible, but the characteristic dark green vein-banding is well shown at first. Later this symptom disappears and the plant becomes a symptomless carrier. Natural infections of *S. nigrum* with this virus in the field are quite common.

Lycopersicum esculentum. Tomato. The tomato plant is readily susceptible to infection with *Solanum Virus 2*, both by inoculation and by the agency of the aphid *Myzus persicae* Sulz. The symptom expression follows the usual sequence of vein-clearing and mottling, but unless carefully watched for, may easily be missed. Clearing of the veins develops after seven to nine days, followed a few days later by the characteristic green banding of the veins. The symptoms are never strongly marked, and as the plant grows they disappear entirely and the tomato carries the virus without symptoms.

Hyoscyamus niger. Henbane. The symptoms induced by this virus upon henbane resemble very closely those produced upon tobacco by the same virus; clearing of the veins develops in seven days, and this is followed shortly after by the usual vein-banding.

Petunia sp. Garden variety. Clearing of the veins is hardly perceptible in this plant, but the darkening of the green colour in the tissues along the veins is quite distinct.

Geographical Distribution and Economic Importance. So far as the British Isles are concerned, *Solanum Virus 2* (potato virus Y) is widely distributed in the south and east of England, and is comparatively common in the south-east of Ireland. In Scotland and in the best potato-growing districts of Ireland this virus is rare. It is very common in different parts of France, and the writer has found it widely distributed throughout the tobacco crops in the Bordeaux district. It is common in many parts of North America, where it is known as the "vein-banding virus." This virus is of great economic importance, and either alone or in combination with *Solanum Virus 1* (potato virus X) is responsible for much loss to the farmer.

Solanum Virus 2 resembles in many ways *Solanum Virus 3*, and

is possibly related to it. Murphy places them together in a group of Y-type viruses.

SOLANUM VIRUS 3. Murphy and M'Kay

Synonyms. *Potato Virus A*, Murphy and M'Kay, 1932; *Super-mild Mosaic Virus*, Quanjer.

The Virus

It is not easy to investigate the physical properties of this virus, because it soon loses infective power and is not readily transmissible by sap-inoculation.

Thermal Death-point. No infections were obtained in a small number of tests with sap heated for ten minutes at 50° C. or higher (5).

Dilution End-point. This is apparently very low, between 1 : 50 and 1 : 100.

Resistance to Ageing. The virus is inactivated in expressed sap after twenty-four hours at room temperatures.

Transmission. The insect vector in the British Isles is the aphid *Myzus persicae* Sulz. (see p. 538) (27), and in North America *Aphis rhamni* (*abbreviata*). The virus is not transmissible from potato to potato by ordinary rubbing with infective sap ; it can, however, be transferred in this way if an abrasive such as fine carborundum powder is added to the sap. It is a curious fact that the virus is sap-transmissible to tobacco by ordinary rubbing, though no doubt the addition of carborundum powder would facilitate transmission in this case also.

Differential Hosts

In many of its reactions this virus resembles *Solanum Virus 2* (potato virus Y), but the fact that there exists no immunological relationship between the two seems sufficient justification for considering them as independent entities. Murphy and Loughnane (34) suggest that each may be the type of a smaller group of closely related or practically identical viruses.

On tobacco, var. White Burley, *Solanum Virus 3* produces a clearing of the veins of the youngest leaves about seven days after infection, and this is followed by a green banding of the veins of the older leaves (see Fig. 68, B). This symptom picture is practically identical with that of *Solanum Virus 2* (potato virus Y) on the same plant, except that in the latter case the symptoms are more pronounced. *Datura Stramonium* is immune to both viruses.

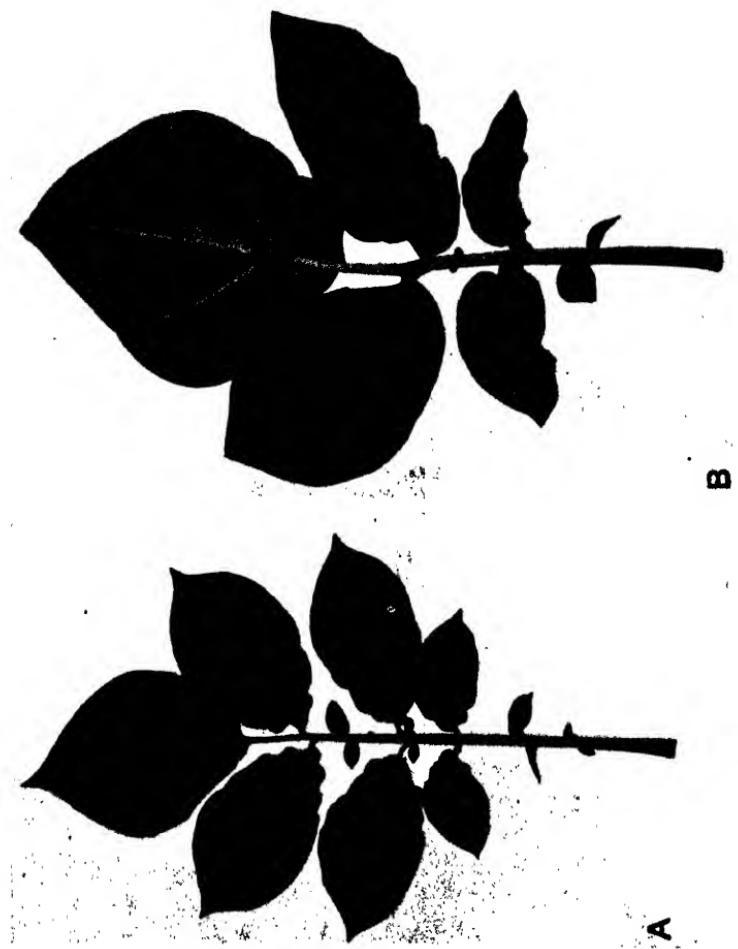


FIG. 62. *Solanum Virus 3* (potato virus A).

- A. Infected President potato.
- B. Infected British Queen potato.
(After Murphy.)

by inoculation, but *Solanum Virus 3* (virus A) can be transmitted to this plant by grafting. The two may be differentiated by inoculation to *Solanum nodiflorum*, on which *Solanum Virus 3* produces no symptoms, while *Solanum Virus 2* (potato virus Y) gives rise to a pronounced veinal mosaic (34).

The combination of *Solanum Viruses 2* and *3* (Y and A) produces a pronounced yellow mosaic in the lower and middle leaves of the potato varieties, Irish Chieftain, Golden Wonder, President and others. *Solanum Virus 3* also gives rise to small brown necrotic flecks throughout the flesh of tubers of the variety Arran Crest.

Diseases in Different Potato Varieties caused by *Solanum Virus 3*

This virus is one of the constituents of the complex disease known as "crinkle," and the symptoms of this disease are described on p. 391.

Irish Chieftain. Plants of this variety containing the virus usually present few or no symptoms. A faint mosaic mottle and vein-clearing accompanied by a slight marginal undulation is frequently seen in the early stages of growth under glass. Later the plants become more normal in appearance and no definite symptoms of disease can be observed. There are no tuber lesions. Most commercial stocks of this variety are infected with the virus.

President (or Paul Kruger). Here the symptoms are a very faint mosaic, without any distortion of the leaf. Following infection, a slight general chlorosis appears on the upper portion of the plant, and on close examination a yellowish mottling can be seen which consists of partially green areas interspersed with normally green ones (see Fig. 62, A).

Arran Victory. In this variety either a faint and fleeting mottle is caused or else the virus is carried without symptoms.

Epicure. A more definite mottle develops in Epicure, taking the form of a distinct yellowing round the veins.

Golden Wonder, Myatt's Ashleaf. Both these varieties carry the virus without symptoms, and it is present in most commercial stocks.

British Queen, Up-to-Date, International Kidney, Kerr's Pink, Rhoderick Dhu, Sharpe's Express and Great Scot. On all these varieties the virus produces a severe disease of top necrosis which invariably results in the death of the plant. The tubers from such plants show severe necrosis, which appears to originate in the

internal phloem, but rapidly embraces all the tissues, so that the eyes are killed and in many cases the entire tuber rots and shrinks (18) (see Fig. 62, B).

Quanjer (41) has defined top necrosis (acronecrosis) on the basis of the internal symptoms of affected plants as "necrosis radiating from only a small percentage of the internal phloem strands, almost never from the external phloem strands, into the surrounding parenchyma, this in turn surrounded by a cork cambium, except in the tender tips, which are soon killed; occurring in foliage, stem and tubers." Externally the disease is seen as a necrotic spotting of the uppermost leaves, which is usually followed by the death of the growing points and the dying back of the plant from the top downwards. The external symptoms are similar to, but differ slightly from, those of the top necrosis produced in Epicure by *Solanum Virus 1* (potato virus X), see p. 344. The disease, however, spreads more slowly and the individual necroses are fewer in number and larger. Internal necroses are found arising in, and spreading from, the phloem in both stems and tubers. Plants in the second year of infection are usually small, brittle, highly necrotic and short-lived (5).

SOLANUM VIRUS 4. Murphy

Synonyms. *Up-to-Date Streak Virus*, Murphy; *Potato Virus B*, Bawden, 1936.

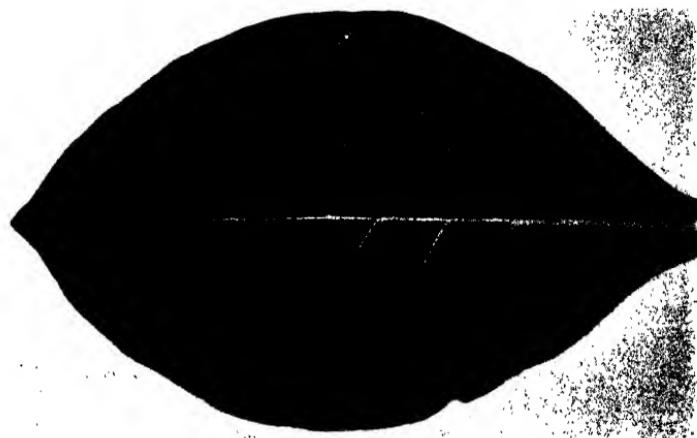
The Virus

This virus appears to be nearly always present in company with *Solanum Virus 1* (potato virus X) in the variety Up-to-Date. It has not yet been isolated from the accompanying virus, but from inoculation tests with the two viruses Bawden (5) considers that its resistance to dilution and ageing is low, since inoculum diluted to 1 : 1,000 or aged for four weeks gives reactions typical of *Solanum Virus 1* (potato virus X) alone. Its thermal death-point seems to be about 70° C. for ten minutes' exposure.

According to Clinch and Loughnane (18), the virus is filterable through an L₃ Pasteur-Chamberland candle and inoculable by needle to *Datura Stramonium* and tobacco. It is transmissible from *Datura* to potato by grafting, but not by needle inoculation. It is also transmissible by needle from Up-to-Date to Arran Crest potato, in which it produces top necrosis.

In a recent paper Dykstra (21) considers that he has obtained

B



A



FIG. 63.
A. *Solanum Virus 4* (potato virus B) on President potato.
B. *Solanum Virus 3* (potato virus A) on tobacco, White Burley.
(After Murphy.)

Solanum Virus 4 alone by the use of a new potato seedling which is resistant to *Solanum Virus 1*.

Diseases in Different Potato Varieties caused by *Solanum Virus 4*

Potato plants of the following varieties have been found naturally infected with *Solanum Viruses 4* and *1* (B and X) : Arran Banner, Arran Consul, American Wonder, Burbank, Bliss Triumph, Duke of York, Earliest of All, Eclipse, Great Scot, Green Mountain, Majestic and Up-to-Date. When infected, these all looked healthy or showed a faint mottle characteristic of infection with *Solanum Virus 1* (X) alone. Di Vernon, International Kidney and Myatt's Ashleaf can also act as carrier of the two viruses. The following varieties are intolerant of the virus combination and react with a top necrosis, i.e., the development of necrotic spots on the youngest topmost leaves and a killing out of the growing points : Abundance, Arran Cairn, Arran Chief, Arran Comrade, Arran Crest, Arran Pilot, Arran Scout, Arran Victory, British Queen, Cattriona, Edzell Blue, Epicure, Golden Wonder, Katahdin, Kerr's Pink, King Edward, May Queen, President (see Fig. 63, A), Rhoderick Dhu and Sharpe's Express. As Bawden (5) points out, since those varieties are infected with both viruses it is not certain whether the top necrosis could be brought about by *Solanum Virus 4* (virus B) alone or whether the condition is dependent upon infection by both viruses. There is some evidence, however, that top necrosis is caused by *Solanum Virus 4* alone, since Dykstra (20), by grafting a potato carrier infected with both viruses to a potato seedling stated to be immune to *Solanum Virus 1* (potato virus X), obtained a source of *Solanum Virus 4* free from the accompanying *Solanum Virus 1*. When transferred to Arran Victory and President this virus alone produced the top necrosis disease.

SOLANUM VIRUS 5. Salaman

Synonyms. *Di Vernon Streak*, Salaman, 1930 ; *Virus C*, Bawden, 1936.

The Virus and Diseases caused by it

Like the preceding virus, *Solanum Virus 5* has not been obtained alone. Salaman, in 1930 (*Nature*, 126), showed that potato

plants of the variety Di Vernon when grafted to Arran Victory and President gave the top necrosis disease in the latter variety only, whereas similar grafts of Up-to-Date containing *Solanum Virus 4* gave top necrosis in both Arran Victory and President. Following up this work, Bawden (5) has found the virus in two stocks of Di Vernon in company with *Solanum Viruses 1* and *2* (potato viruses X and Y) and in one stock of Monocraat together with *Solanum Virus 1*.

Solanum Virus 5 has not been transmitted to any plants by sap inoculation. No local lesions are produced as a result of inoculation to President potato. On American potato varieties such as Burbank, Bliss Triumph, Irish Cobbler and Earliest of All (see Fig. 64, A and B), the virus produces a severe top necrosis, consisting of a streaking of the stem and petioles and numerous small circular spots on the foliage (21). Plants of *Datura Stramonium*, tobacco and tomato when inoculated and then grafted to President did not give top necrosis. Further differences between *Solanum Viruses 4* and *5* are seen in their reactions on various potato varieties. *Solanum Virus 5* produces top necrosis in Arran Consul, Arran Banner, Duke of York, Eclipse, Majestic and Up-to-Date, all of which carry *Solanum Virus 4* without symptoms, as well as in Arran Crest, Arran Pilot, Arran Cairn, British Queen, Epicure, King Edward, President and Sharpe's Express. *Solanum Virus 5* is believed to be carried by Arran Victory, Arran Chief, Great Scot and Rhoderick Dhu, for when plants of these varieties are grafted with Di Vernon they show symptoms typical only of infection with the other viruses present (5).

Recently Dykstra (21) has grafted scions from infected Di Vernon plants to several plants of the varieties Burbank, Bliss Triumph, Irish Cobbler and Earliest of All. In nearly every case current season symptoms developed which were characterised in these varieties by a severe top necrosis, consisting of a streaking of the stem and petioles and by numerous small circular necrotic spots on the foliage. The second generation symptoms of this disease on these varieties as observed on plants growing under cages were manifested by a slight mottling of the foliage without any noticeable dwarfing and in many cases without any indication of necrosis. In a few plants of Irish Cobbler circular necrotic spots were found on the lower leaves. This virus is considered by Dykstra to be distinct from any of the known potato viruses occurring in America.

SOLANUM VIRUS 6. Murphy

Synonyms. *President Streak Virus*, Murphy, 1927; *Potato Foliar Necrosis Virus*, Bawden, 1934; *Potato Virus D*, Bawden, 1934.

The Virus

It is probable that this virus is a strain of *Solanum Virus 1* (potato X virus), but owing to the difference in the symptoms it produces on certain potato varieties from the symptoms produced by *Solanum Virus 1* and to certain differences in its physical properties it is dealt with here as a separate entity.

Resistance to Various Chemicals. The virus is completely inactivated by 60 per cent alcohol acting for one hour, but not by 50 per cent. By phenol and formalin it is destroyed by solutions of 4 per cent, but not by 2 per cent also acting for one hour. Saponin at 1 : 20 has no effect on infectivity. Psychosyn, a lytic principle from brain tissue, at 1 : 20, inactivates the virus in purified suspensions, but has no effect on the infectivity of crude expressed sap (4).

Serological Reactions. This virus reacts with antiserum prepared against *Solanum Virus 1* (potato virus X), and this is further evidence of the close relationship existing between the two viruses.

Thermal Death-point. Ten minutes' exposure at 67° C. inactivates the virus.

Dilution End-point. With juice derived from young actively growing tobacco plants infected with this virus infection can usually be obtained at dilutions of 1 : 5,000, but not at 1 : 10,000. From old infected tobacco plants the effect of dilution is much more marked and the end-point is usually about 1 : 1,500. With sap derived from infected potatoes no infections have been obtained at dilution greater than 1 : 2,000, and from mature potato plants the dilution end-point may be as low as 1 : 750.

Resistance to Ageing. At a temperature of 25° C. the infectivity of the virus rapidly decreases, and it is completely inactivated in six or seven days. At 10° C. the virus is still infective after twelve days' storage, and at 1° C. it has been found to be infectious after a month, but not after six weeks.

Filterability. The virus in a kieselguhr filtrate passes Chamberland candles L₁, L₃ and L₅, though the virus content of the L₅ filtrates is low.

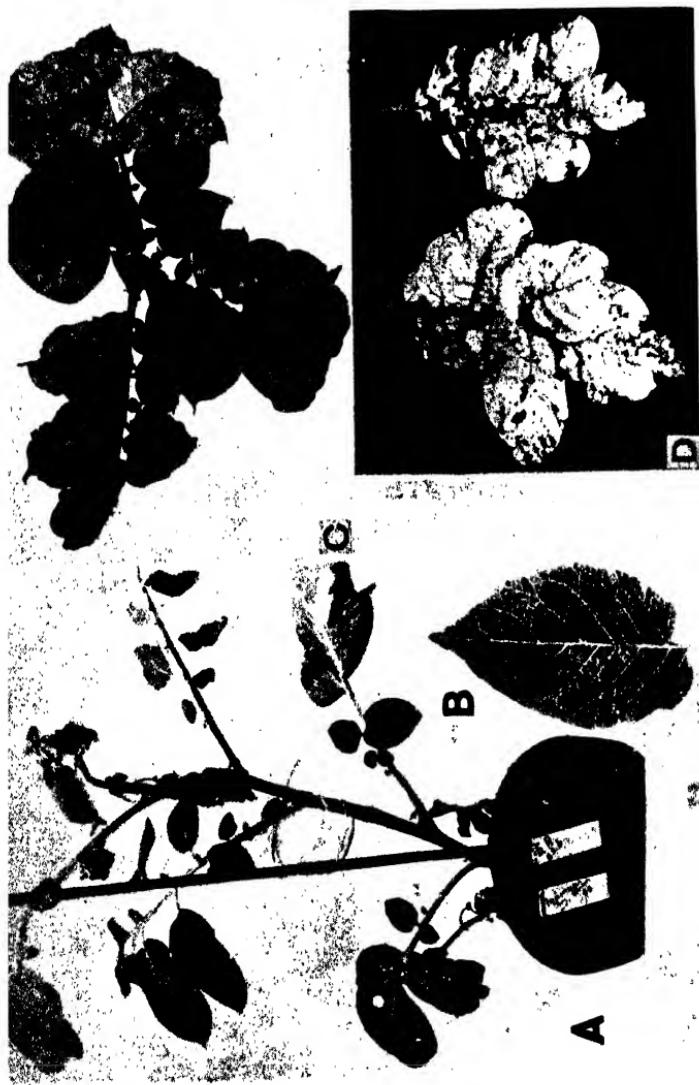


FIG. 64. A and B. Current season symptoms of top necrosis produced by *Solanum Virus 5* (potato virus C).

- A. On potato Earliest-of-All. Note necrotic spotting on the foliage and petioles.
- B. On potato Liss Cobbler. Note necrotic spots.
- C. President potato infected with *Solanum Virus 6* (potato virus D).
- D. Leaves of President potato infected with *Solanum Virus 6* (infra-red plate). (A and B, after Dykstra; D, after Bawden.)

Transmission. The virus is sap-transmissible; the insect vectors, if any, are not known.

Diseases in Various Potato Varieties caused by *Solanum Virus 6*

Arran Victory. When the virus is transmitted to this variety by sap-inoculation local lesions frequently form on the inoculated leaves, but if the virus is transmitted by grafting then no local lesions are produced. The lesions appear eight to ten days after inoculation, and consist of black necrotic spots which penetrate the thickness of the leaf. They are usually more or less circular, brittle to the touch and after a time tend to fall from the surrounding leaf tissue, giving a "shot-hole" appearance. They are extremely localised and once formed show little tendency to spread.

The first systemic symptoms appear about seventeen to nineteen days after infection, and consist of interveinal necrotic blotches on the under surfaces of those leaves occupying an intermediate position on the stem. These necroses, which are greyish in colour and soft and damp to the touch, spread rapidly, coalesce and ultimately affect the whole leaf, causing it to wilt and finally to fall. The advance of the disease is acropetal and, starting with the intermediate leaves, the wilting and falling of the leaves spread upwards. These primary symptoms are then followed by a secondary and less severe stage of the disease. The upper leaves show a pronounced and rather blotchy interveinal mosaic. In addition, spotty black interveinal necroses appear which may cause acute local deformity. These necroses differ in appearance from those of the primary stage of the disease and more closely resemble the local lesions. In this phase of the disease the wilting and leaf fall are arrested and new growth shows only the interveinal mottle and scattered black necroses. The tubers from such affected plants, though small, appear healthy, and on planting sprout normally.

Arran Victory plants, in the second year of infection with this virus, show symptoms similar to the secondary phase of the disease in a current season infection. In the field such plants are dwarfed, mature very early, and set only a few small tubers. The leaves are much under-developed, extremely harsh and brittle, and covered with many small black necroses. The mottle is usually less well developed than on plants grown in the glasshouse. In addition, the leaves are often definitely bronzed or

"rusty," a condition which seems to be peculiar to virus-infected potato plants grown in the open.

Histopathology

Histological examination of the leaves of affected plants shows that the necrosis originates in the parenchymatous cells abutting on the small vascular bundles found between the main veins. From this region the necroses spread outwards to the epidermis and along the length of the leaf, affecting both palisade cells and spongy parenchyma. No necrosis has been found in the stems and tubers of Arran Victory plants infected with *Solanum Virus 6*.

In leaves showing the mosaic mottle, the chlorotic areas are noticeably thinner than the green areas and the palisade cells of the former are under-developed. No intracellular inclusions, or X-bodies, have been observed associated with this disease.

President. The disease produced by *Solanum Virus 6* on President potato differs only slightly from that described for Arran Victory. In the secondary stage the interveinal mosaic is more pronounced in President and necroses are fewer, though of a similar type. In the second year of infection President plants are not usually so severely affected as Arran Victory. The interveinal mottle is much brighter, but the black necroses are fewer and do not cause such acute deformity. The flowering of President plants seems to be affected by infection with this virus and very few flowers, if any, are produced per plant (see Fig. 64, C and D).

Other Potato Varieties. In addition to Arran Victory and President, the following varieties have been found to show similar symptoms when infected with *Solanum Virus 6*: Arran Cairn, Arran Chief, Arran Comrade, Arran Pilot, British Queen, Edzell Blue, Katahdin, Kerr's Pink, May Queen, Rhoderick Dhu and Sharpe's Express.

Epicure. On grafting the virus to Epicure potato, the first symptom is an extensive necrotic spotting of the uppermost leaves surrounding the growing point. The necroses coalesce, kill out the growing points and the plants die back from the top. The disease produced in Epicure, therefore, is a top necrosis exactly similar to that produced in the same variety by *Solanum Virus 1* (potato virus X). Internal lesions are found in the stems of such affected plants. These originate in the phloem, usually in the internal phloem, and spread from there to all other tissues. Tubers set by these plants appear normal, but may become necrotic during storage. Such necrotic tubers, when planted, either fail to grow

or give rise to plants which never reach a height of more than a few inches, are covered with fine necroses and quickly die. The varieties Arran Crest and King Edward react similarly to infection with *Solanum Virus 6*.

Up-to-Date. This variety also develops a top necrosis when infected with the virus, but the disease is of a slightly different character. The first necroses appear on the veins of the uppermost leaves or at the top of the main stem and consist of dark brown or black stripes. From the veins the necroses pass into the parenchyma and the whole apex of the plant is killed. The progress of the disease from this point depends largely on the stage of growth of the plant. If it is young and fleshy it usually dies back from the top and the whole plant is killed. If older and woody, the destructive necroses are frequently restricted to the actual growing points. In such plants the necroses cause acute twisting and crinkling of the uppermost leaves. Internal necroses are found in the stems and petioles and originate in the phloem. The bundles chiefly affected in this case, however, are those of the external phloem, and from these the necroses spread outward through the cortex, appearing externally as black stripes. Other potato viruses responding similarly to infection with *Solanum Virus 6* are Arran Consul, Majestic and Duke of York.

Abundance. The virus produces a top necrosis also in this variety, but again the disease is of a slightly different type. Necroses first appear on the leaves immediately below the apex, and spread from these into the growing point, which is then killed. The spread of the disease becomes basipetal, i.e., towards the base of the plant, which dies back from the top. Internal lesions develop in the stem, where they originate in the phloem and pass out to the epidermis. Second-year plants are much dwarfed and are at first smothered with numerous small black necroses. These coalesce, killing out large areas of the leaves, which shrivel and fall and the whole plant soon matures.

Symptomless Carriers of *Solanum Virus 6*

The following potato varieties are considered by Bawden (4) to carry the virus without symptoms :

Arran Banner, Arran Scout, Champion, Eclipse and Di Vernon.

Diseases in Other Solanaceous Plants caused by *Solanum Virus 6*

Nicotiana tabacum. Tobacco, var. White Burley. Faint chlorotic lesions develop on the inoculated leaves of tobacco five

to seven days after inoculation. The systemic symptoms, which appear about twelve days after infection, take the form of chlorotic spots on intermediate leaves. The chloroses increase in size and frequently assume a ring-like form. The spots and rings are always most definite on the intermediate leaves and the disease is never severe. As the plants grow the symptoms tend to fade and a few weeks after infection the symptoms may have completely disappeared.

N. glutinosa. On this species the disease produced is similar to but much less severe than that on tobacco. It consists of a very faint chlorotic spotting on the intermediate leaves which persists for a few weeks, after which the plant may regain a normal appearance.

Lycopersicum esculentum. Tomato, var. Kondine Red. On tomato the virus produces a very faint but general interveinal mosaic.

Datura Stramonium. The majority of plants of this species appear to carry the virus without symptoms. Occasionally, however, infected plants have developed a mild chlorotic spotting which appears about twelve days after inoculation. The spots persist for about a fortnight, after which they fade and the plants again appear normal (4).

SOLANUM VIRUS 7. Salaman and Le Pelley

Synonyms. *Paracrinkle Virus*, Salaman and Le Pelley, 1930; *Potato Virus*, E. Bawden (Smith, 1933).

The Virus and its Transmission. As the virus is not sap-inoculable, there is no information available on its physical properties. This virus can only be communicated by stem-grafting, and the insect vector, if such exists, is not known. The virus does not appear to spread naturally in the field. The distribution is throughout the variety King Edward, but in no other variety.

Diseases in Different Potato Varieties caused by Solanum Virus 7

King Edward. Strictly speaking, no disease is caused in this variety by the virus because it is carried without symptoms and does not appear to affect the health of the plant in any way. It is an interesting fact that apparently all stocks of the King Edward potato are naturally infected with the virus, and plants of this variety free of the virus have not been observed. The

varieties President, and possibly Champion also, when infected artificially, carry the virus without symptoms (47).

Arran Victory. The first symptom is the development of a single bright blotchy patch of chlorosis on the junction of the larger leaf veins, accompanied by local puckering and deformity allied with a characteristic waving of the leaf edge. Clearing of the veins is usually present in a variable degree. The blotchy patches increase in number with a corresponding increase in deformity of the leaves. Growth of the leaflets is more or less completely arrested, and by reason of the waving, deformity and rugosity they appear smaller than their actual area warrants. Necrotic spots are common on the surface of the leaf and short brown streaks on the veins of the under surface, as well as on the petioles and stems. In the first season, the symptoms are chiefly confined to the younger upper growth and the plant is in consequence not stunted to a great degree. In subsequent seasons the disease is much more serious, the stems are numerous, short and brittle, the leaves are clumped together and grossly deformed and the necrotic lesions are intensified (see Fig. 70, A).

Arran Victory plants infected with this virus when grown in the field are of the "curly dwarf" type; and are often not more than 6 or 9 inches high.

It will be well to emphasise here the difference in symptoms caused by *Solanum Virus 7* (paracrinkle virus) and those of the crinkle disease which is a composite infection (see p. 391). Clearing of the leaf veins as an early symptom is common to both. Mottling in crinkle begins at the junction of main veins and extends along them ; it is usually not very extensive or bright and tends to fade rapidly. With the former virus the mottling also begins at the junction of veins, but rapidly develops into a more or less circular, blotchy and bright marking. Deformity may be absent in crinkle and is never very pronounced, but in the other case deformity is an important feature and is often so extreme as to render the leaf unrecognisable (47). It should be realised, perhaps, that these symptoms of paracrinkle are in a sense artificial and are never seen in nature.

Arran Chief. In this variety the symptoms are very similar to those in Arran Victory, but slightly less severe. Plants in their second year of infection develop into the same type of curly dwarf when grown out of doors.

Arran Comrade, Majestic, Great Scot. These three varieties react with mosaic rather than crinkle symptoms.

The first-named produces only a mild mosaic, while in Majestic a rather more severe disease develops. The reaction of Great Scot to the virus is rather variable and the symptoms may vary from a severe mosaic to almost no symptoms. The mosaic starts, like the crinkle, with a clearing of the veins, followed by a development of paler areas somewhat like interveinal mosaic. Sharpe's Express and Epicure appear to be resistant to infection with *Solanum Virus 7*. This virus seems to be distinct from American potato viruses. On the foliage of American varieties of potatoes it produces large irregularly shaped mosaic-like blotches. Under cages in the field pinpoint-like necrotic spots, in addition to mottling, develop in the leaves of infected Burbank plants (21).

Diseases in Other Solanaceous Plants caused by *Solanum Virus 7*

Datura Stramonium. Plants of this species can be infected by grafting with diseased scions, but not by inoculation. Symptoms appear in thirteen to fourteen days as a soft diffuse, rather large type of mottle beginning at the apex of the leaf. Later numerous small bright yellow spots develop, more or less clustered towards the leaf apex. New leaves arising later do not develop these spots, but present a mottling less patchy than the primary one and more distinctly interveinal.

Nicotiana tabacum. The tobacco plant appears to be immune from infection with this virus (47).

SOLANUM VIRUS 8. Clinch, Loughnane and Murph.

Synonyms. *Potato Virus F*; *Tuber Blotch Virus*, Clinch, Loughnane and Murphy, 1936; *Pseudo-netnecrosis Virus*, Quanjer, 1931; *Monocraat Virus*.

The Virus

The virus has been found in nature in the varieties Up-to-Date and Monocraat, and is carried almost symptomlessly by many varieties. It probably occurs widely in connection with tuber necrosis, of which it is a principal cause. It appears to be closely related to *Solanum Virus 9*, as evidenced by physical properties and host reactions, but is differentiated, amongst other methods, by absence of yellow spotting on tomato and *Solanum dulcamara* and more severe tuber symptoms.

Thermal Death-point. The virus is inactivated by ten minutes' exposure to temperatures of 68° to 65° C.

Dilution End-point. This is probably about 1 : 100.

Resistance to Ageing. Two to three days at a temperature of 15·5° C.

Filterability. Passes an L₁ Pasteur-Chamberland filter, but not L₃ or L₅.

Transmission. The virus is easily sap-inoculable to potato as well as other host plants, and the insect vector is the aphid *Myzus persicae* Sulz. (see p. 538). There is an interesting fact associated with the transmission of this virus by the aphid, *i.e.*, that it seems necessary for *Solanum Virus 3* (virus A) also to be present to enable the insect to pick up the former virus. No explanation of this association of the two viruses with insect transmission is available at the moment, but insect transmission of *Solanum Virus 8* alone has not been secured (14).

Differential Hosts

Solanum nodiflorum. About eight days after inoculation slight pale local lesions with brown or purple margins develop on the inoculated leaves. Three to five days later, if the virus becomes systemic, vein-clearing sets in on the leaves next above, developing acropetally, and showing most characteristically on the fourth or fifth leaf from the top. The plants stop growth, and turn a dull dark green. The lowest leaves rapidly become yellow and drop, and the next higher leaves develop brown, purple-fringed spots or a generalised rusty-purple colour, and may wither from the top backwards, and drop also. In some cases the virus does not become systemic, especially in cold weather, which is unsuitable for the plant (see Fig. 65, B).

Capsicum annuum. In capsicum the symptoms are similar to those detailed above, and are usually conspicuous, consisting of pale areas on the inoculated leaves. These leaves become bronzed, curl down, and fall off. Vein-clearing meanwhile appears on the next higher leaves, followed by mosaic, distortion and leaf-drop, and though the leaves become progressively infected the plants survive indefinitely.

Diseases in Different Potato Varieties caused by *Solanum Virus 8*

Dunbar Yeoman and Epicure. These two potatoes are the best diagnostic varieties. In a graft transmission of the virus the

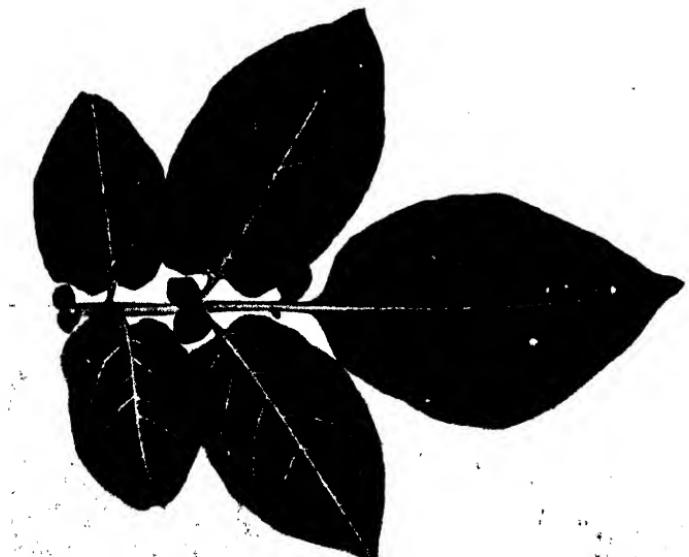


FIG. 65. *Solanum Virus 8* (tuber blotch virus).
A. On potato *Monocrota*, showing a few bright yellow spots on the lower leaves.
B. On *Solanum nodiflorum*, showing large pale spots with brown and purple margins.
(After Murphy.)

latter shows a characteristic general yellowing of the leaves and stems, resulting in leaf-drop and premature death. Dunbar Yeoman shows a bright yellow mottling or severe scorching of the lower leaves or both effects in succession. The necrosis begins as a burn of the tip or margin of a terminal leaflet, and later of the other leaflets, of a middle or lower leaf, rapidly extending inwards along the interveinal areas as a pale green or rusty wilt, and progressing acropetally on the plant. The leaves look as though scorched by fire, curve downwards, gradually turn rusty or yellow and die back, hang or drop. Occasionally there may be slight lesions in the collenchyma of the stem. Dunbar Yeoman also shows the most pronounced secondary symptoms in the field. These consist of a non-necrotic yellow variegation of the tips, margins and interveinal areas of the lower and middle leaves, approximating in brightness to the symptoms produced by *Solanum Virus 9* (potato aucuba mosaic virus) (14). Necrosis of the tubers is a characteristic symptom of attack by this virus and necrosis is particularly severe in the tubers of the varieties President and Roode Star.

Potato varieties may be grouped as follows according to their reactions to this virus. The following varieties show definite primary symptoms, typically of rusty wilt on the lower leaves : Dunbar Yeoman, Epicure, Majestic, Sharpe's Express, British Queen. The following usually show slight symptoms, typically of transient interveinal rust or yellow spots on the lower leaves : President, Arran Victory, Champion, Arran Banner, Arran Consul, Great Scot, Di Vernon, Duke of York, Monocraat (see Fig. 65, A). The following varieties usually show no symptoms : Alpha, Arran Cairn, Arran Crest, Ballydoon, Dunbar Cavalier, Kerr's Pink, King Edward, Roode Star, Eigenheimer. The following varieties, when already containing *Solanum Virus 1* (virus X), develop interveinal mosaic when infected with *Solanum Virus 8* : Up-to-Date, Bliss Triumph, Green Mountain. The following varieties, when already containing *Solanum Virus 3* (virus A), develop a bright yellow mosaic when infected with *Solanum Virus 8* : Irish Chieftain, Golden Wonder, Bohm's Allerfruheste Gelbe.

The secondary symptoms, when present, always consist of a few yellow spots on the lowest leaves, which are most evident in the field early in the season, but are easily overlooked unless accentuated by the presence of *Solanum Virus 3*.

The production of tuber necrosis (sometimes termed "pseudo-

netnecrosis") is one of the most characteristic and economically important features of this virus. Severe necrosis, usually visible on the surface, is produced on the tubers of the following varieties : President, Arran Consul, Majestic, Golden Wonder, Great Scot, British Queen, Dunbar Cavalier, Dunbar Yeoman, Bohm's Allerfruheste Gelbe, Roode Star, Zeeland Blue. The following generally show internal necrosis : Arran Victory, Irish Chieftain, Up-to-Date, Duke of York. The following varieties have shown no symptoms in the tubers : Arran Crest, Arran Banner, Alpha, Kerr's Pink, King Edward, Eigenheimer, Epicure, Di Vernon, Ballydoon. The variety Green Mountain appears to show a superficial browning of the skin which does not penetrate the flesh (Murphy *in litt.*).

Solanum Virus 8 is inoculable to, but produces no symptoms on, *Nicotiana tabacum*, *N. affinis*, *N. glauca*, tomato, petunia, *Datura Stramonium* and *Solanum racemosum*.

SOLANUM VIRUS 9. Murphy and Quanjer

Synonyms. *Potato Aucuba Mosaic Virus*, Murphy and Quanjer ; *Potato Virus G*, Clineh, Loughnane and Murphy, 1936 ; *Non-infectious Chlorosis Virus*, Murphy.

The Virus

The disease caused by this virus was first described and illustrated by Quanjer in Europe, and by Murphy in America, and is of general distribution throughout Great Britain, Europe and North America in occasional plants of many varieties. It has been shown that the American and European diseases are probably identical. The consequent reduction in vigour and yield is not considerable, but more harm is done by the resulting tuber necrosis in several potato varieties. *Solanum Virus 9* is related to *Solanum Virus 8* (tuber blotch virus).

Serological Reactions. Chester (11) has shown that this virus is antigenically unrelated to other viruses affecting the potato.

Thermal Death-point. Ten minutes' exposure at a temperature of 65° C. inactivates the virus.

Dilution End-point. Few positive infections are given with virus sap diluted 1 : 200 and none at dilutions of 1 : 500.

Resistance to Ageing. The limit of survival of the virus when stored at 15° C. in the absence of light is less than four days.

Filterability. When filtered through Pasteur-Chamberland

candles in a paper-pulp filtrate, the virus has been found to pass an L₁ filter, but not L₃ and L₅.

Transmission. The virus is easily transmitted by sap-inoculation not only to potato, but to certain other Solanaceous hosts as well. The insect vector is probably *Myzus persicae* Sulz. (see p. 538).

Differential Hosts

Potato Irish Chieftain. The reaction of this potato variety to the virus is exceptional, and a brilliant yellow mottle develops all over the plant, and not only on the lower leaves as in some potato varieties.

On tomato (var. Kondine Red) *Solanum Virus 9* produces small rounded yellow spots which are confined to the lower leaves. These symptoms show best on soft young plants.

Diseases in Different Potato Varieties caused by *Solanum Virus 9*

President, Early Regent, Majestic, Champion. On these four varieties bright yellow spots develop on the lower leaves of infected plants. The tubers are necrotic except in the case of Early Regent.

Epicure, Arran Crest, Arran Banner. These varieties show a brilliant and extensive yellow spotting on the lower and middle leaves. There is no necrosis of the tubers.

Great Scot, Dunbar Yeoman. These two show similar foliage symptoms to Epicure, but the tubers are necrotic.

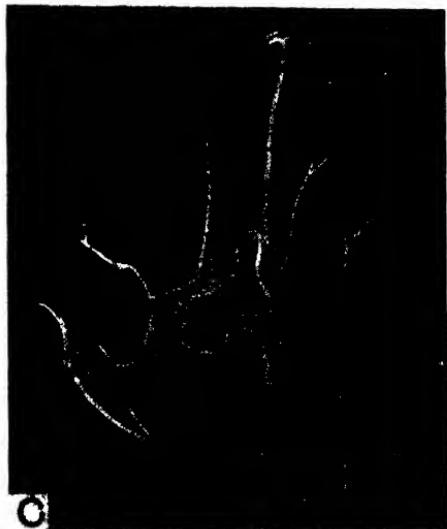
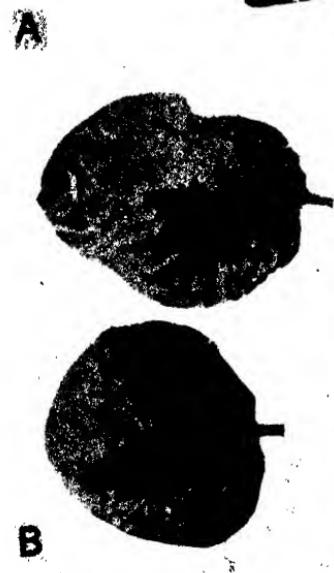
Arran Victory. This variety develops large yellow blister-like spots on the lower leaves without tuber necrosis.

British Queen. Infected plants of British Queen develop first a wilting and "drying-out" of the tips of the lower leaves. This is followed by a pronounced yellow mottle. The tubers are necrotic.

Roode Star. Symptoms on Roode Star are variable; there is a mild and transient diffuse yellowish mottle which is confined

FIG. 66.

- A. Potato, var. International Kidney, affected with *Solanum Virus 9* (potato aucuba mosaic virus).
- B. Leaves of potato plant affected with *Solanum Virus 10* (potato calico virus).
- C. Tuber from potato plant affected with *Solanum Virus 15* (potato witch's broom virus): note spindling sprouts. (B, after Porter; C, after McKay and Dykstra.)



to the bottom leaves or else a yellow blistering at the tips of these leaves. Necrosis may occur in the tubers, but this symptom is erratic in appearance.

Up-to-Date. In plants of Up-to-Date already carrying *Solanum Virus 1* (potato virus X), this virus produces a bright yellow mottle on the lower leaves without necrosis in the tuber.

Irish Chieftain. As already mentioned, infected plants of Irish Chieftain exhibit a brilliant yellow mottle which reaches to the top of the plant. The unusual brilliance of the symptom is considered by Clinch, Loughnane and Murphy to be due in part to the action of *Solanum Virus 3* (virus A), which is usually present in this variety. The tubers are not necrosed.

Histopathology. The cells of the green areas in affected potato leaves appear normal, with well-developed green plastids containing small starch grains. In the transitional zone the plastids are of a pale green colour, more granular in appearance, and sometimes irregular in shape. In the white areas the chloroplasts are almost colourless and contain abnormal quantities of starch. In many cells they may be in a degenerating condition, displaying large vacuoles from which the starch has disappeared. Intracellular inclusions, or X-bodies, have not been observed in association with this virus (12).

In the tuber, necrosis frequently occurs in the parenchymatous cells of both cortex and pith and is usually visible externally as irregularly shaped brown patches which later develop into sunken dry brown areas. It begins first towards the heel end of the tuber, and does not attack the vascular tissue or eyes. The necrotic patches consist of groups of misshapen dead cells with brown granular contents and swollen brown and disintegrating walls, surrounded by a zone of translucent tissue containing little or no starch in which incipient cork formation is observed. The centre of the spot is occupied by a small group of cells filled with large starch grains. The necrosis develops during storage, and is favoured by darkness and high temperatures and particularly by a turgid condition (14).

Other Solanaceous Host Plants. *Datura Stramonium*, tobacco, var. White Burley, and petunia (var. Rosy Morn) are all susceptible to the virus by sap-inoculation, but they show no symptoms. The reaction of the tomato plant to this virus has already been described. On *Solanum nodiflorum* and *Capsicum annuum* the symptoms are similar to those produced by *Solanum Virus 8* (tuber blotch virus).

SOLANUM VIRUS 10. Hungerford

Synonym. *Potato Calico Virus*, Porter, 1931.

The Virus

It is probable that this virus has some affinities with *Solanum Virus 9* (potato aucuba mosaic virus), since the respective diseases in the potato are very similar though not quite identical. The virus has only been recorded from North America, where it appears to be widely distributed (39).

Transmission. The virus is sap-inoculable and can also be transmitted by tuber grafts, but only with difficulty. The insect vector does not appear to be known.

Filterability. Attempts to produce infection with filtered sap from diseased potato plants have been unsuccessful.

Disease caused by Solanum Virus 10

Potato, var. White Rose. Some of the leaflets of infected plants growing in the field become irregularly spotted or blotched. These areas do not become necrotic, rather they appear devoid of chlorophyll and generally assume a bright brilliant yellow, yellowish-white, or grey colour (see Fig. 66, B). The spots are not always interveinal and may occupy as much as 95 per cent of the leaflet area, being, as a rule, irregularly scattered. As a probable result of chlorophyll deficiency, plants infected when young seldom attain normal size.

The young leaflets of healthy plants which have been artificially inoculated with unfiltered juice develop symptoms practically identical with those observed in the field; but, in addition to yellowing, the lower leaflets may become necrotic at the tip, crinkled or ruffled, and in some instances slightly stiffened. The minimum incubation period is about fifteen days.

SOLANUM VIRUS 11. Schultz and Folsom

Synonyms. *Leaf-rolling Mosaic Virus*, Schultz and Folsom, 1928; *Potato Virus 7*, J. Johnson's classification.

The Virus and its Transmission. There seems to be no information upon the properties of this virus; it is difficult to transmit by sap-inoculation. According to Schultz and Folsom (51) the virus is aphid-borne.



FIG. 67.

- A. Potato Earliest of All infected with *Solanum Virus 11* (leaf-rolling mosaic virus).
- B. Tubers from Irish Cobbler potato plant infected with *Solanum Virus 12* (potato spindle tuber virus) : *h*, healthy ; *d*, diseased.
(B, after D. J. MacCleod.)

**Diseases in Different Potato Varieties caused by
*Solanum Virus 11***

Arran Victory. The symptoms on this variety are characterised by a diffused mottling and rolling of the leaves. The disease can be distinguished from leaf-roll, *Solanum Virus 14*, by the absence of distinct rolling, dwarfing or rigidity and of chlorosis.

The mottling of the leaflets of infected plants is diffused and resembles the type found in the complex disease, rugose mosaic (see p. 394). The leaves generally show an upward rolling, but the leaves are flaccid and resemble the type of rolling found in plants affected with *Rhizoctonia* or blackleg. There appears to be a distinct difference in the severity of the symptoms in different potato varieties. The rolling and mottling are almost masked in the American varieties Burbank and Irish Cobbler. In some other American varieties, like Earliest of All (see Fig. 67, A), Idaho Rural, Bliss Triumph, Green Mountain and White Rose, the leaves show a distinct rolling, and the mottling is generally distinct, although under high temperatures it is partly or completely masked (28).

SOLANUM VIRUS 12. Schultz and Folsom

Synonyms. *Potato Spindle-tuber Virus*; *Potato Virus 8*, J. Johnson's classification; *Potato Marginal Leaf-roll Virus*, Fernow and Quanjer, 1933.

The Virus

Thermal Death-point. The virus is inactivated by ten minutes' exposure to a temperature between 60° and 65° C.

Dilution End-point. The virus begins to lose infectivity after dilutions of 1 : 1,000 and gives no infection after dilutions of 1 : 10,000.

Resistance to Ageing. There is no exact information on this point, but what evidence does exist suggests that the virus in expressed sap is rapidly inactivated on standing.

Partial Desiccation in Infected Foliage. The virus appears to be more resistant in dried host tissue than in extracted sap.

In experiments conducted by Goss (28) infection was obtained with tissue that had been one week drying, but not after it had been kept seventeen days or longer.

Transmission. The virus is sap-inoculable and can be transmitted by core-grafts in the tubers. It is also transmitted by

rubbing together the cut faces of infected and healthy tubers and by means of a contaminated tuber-cutting knife. The insect vectors of this virus appear to be both numerous and variable. Schultz and Folsom (51) find that aphides, probably *Myzus persicae* and *Macrosiphum gei* (see pp. 538 and 532) are the vectors, and Goss (28) states that grasshoppers (*Melanoplus* spp.), flea beetles (*Epitrix cucumeris* and *Systema tenuata*), the tarnished plant bug (*Lygus pratensis*), the larvae of the Colorado beetle (*Leptinotarsa decemlineata*) and the leaf beetle (*Disonycha triangularis*) are all capable of transmitting the virus.

Diseases in Different Potato Varieties caused by *Solanum* Virus 12

This disease occurs most commonly in the United States of America and Canada. In affected potato plants of the Green Mountain variety the shoots are more erect and spindling than in the normal plant. The leaves are smaller, more erect, and early in the season are somewhat darker green with more rugosity, that is, with the leaf surface raised between the veins, thus decreasing the smoothness and flatness of the leaf. Later in the season the leaves are even more dwarfed, but the rugosity is not so marked. The mid-ribs of lateral leaflets occurring at the top of seriously affected plants are sometimes inwardly curved so that these leaflets tend to overlap each other and the terminal leaflet as well, giving the plant what is popularly called a "bow-legged" appearance. The main petiole is often more slender than normal and may become slightly brittle (29). The most striking effect of the virus is on the tubers, which are made spindling, long and cylindrical, with a more irregular or bumpy outline, more spindle-shaped or tapering ends, and more conspicuous eyes (see Fig. 67, B). Usually the skin of a "spindle" tuber is smoother and more tender, and in the spring the flesh cuts more easily. Occasionally tubers from infected plants occur in which the general short wide shape of healthy tubers is retained, but the skin, eyes, ends and regularity of outline are still affected. In the varieties Irish Cobbler and Bliss Triumph the tubers of affected plants present a difference in skin colour which becomes browner and duller in the former variety and lighter pink and somewhat blotchy in the latter variety. In Irish Cobbler also the effect on the tuber shape is more pronounced than in Bliss Triumph. In the variety Spaulding Rose (Northern Red or King) the tuber skin is made lighter in colour than normal, while the change in the leaves is less than in Green

Mountain. In the variety Rural New Yorker the tuber skin is made smoother, as in Green Mountain, but the tubers are not changed so much from flat to cylindrical (22). According to MacLeod (29) tubers from affected plants of different varieties show protruding " brows " associated with the shallow conspicuous eyes.

The English variety President has been experimentally infected with the virus by Murphy and McKay (36). The infected plants were upright and considerably dwarfed, about 6 inches high, with a red roll in the top like primary leaf-roll (*Solanum Virus 14*) and marked by the same peculiar dying of the leaves from the tip backwards and from the base of the plant upwards. Primary necrosis was also produced on the President foliage, but this may have been due to some other accompanying virus.

SOLANUM VIRUS 13. Schultz and Folsom

Synonyms. *Potato Unmottled Curly Dwarf Virus*, Schultz and Folsom, 1923, and Goss, 1930; *Potato Virus 9*, J. Johnson's classification.

The Virus

The virus causing this disease is very similar to the foregoing, but there seems to be sufficient evidence for considering the two to be separate entities.

Thermal Death-point. The virus is inactivated by exposure to temperatures between 75° and 85° C.

Dilution End-point. This virus, according to Goss, is less tolerant of dilution than *Solanum Virus 12*. He found that there was a drop from 60 per cent infection with a 1 : 100 dilution to 10 per cent with a 1 : 1,000 dilution.

Resistance to Ageing. There is little exact evidence on this point, but the virus appears to lose infectivity rapidly after extraction in the sap of infected plants.

Transmission. The virus is rather easily inoculable by the sap, and is also transmissible by contact of the cut surfaces of the seed tubers and by the tuber-cutting knife. The insect vectors are stated by Goss to be the same as for the preceding virus, with the exception of the leaf beetle (*Disonycha triangularis*) and with the addition of the leafhopper *Euscelis exitiosus*.

Disease caused by Solanum Virus 13

Green Mountain. In this variety the symptoms consist of pronounced dwarfing, spindliness, dark green colour of the foliage

early in the season, wrinkling, rugosity, slight ruffling, curling, some rolling, uprightness, brittleness, burning, somewhat premature death and spindling, gnarled and cracked tubers. The tuber-cracking may appear, without the spindliness, as a symptom of current season infection. On the whole the current season symptoms are more pronounced in the case of this virus as compared with *Solanum Virus 12* and help to differentiate the two viruses. This is especially true of the tuber symptoms. If infection occurs at the time of tuber formation, the longitudinal cracks are more pronounced, and the tubers, while large and well coloured, are often misshapen with abnormal eyes. This cracking of the tubers, together with their bumpy irregular shape, is characteristic of plants infected with *Solanum Virus 13* (un-mottled curly dwarf virus). Plants infected at the same age with *Solanum Virus 12* (spindle tuber virus) would not show symptoms till much later.

SOLANUM VIRUS 14. Appel and Quanjer

Synonyms. *Potato Phloem-necrosis Virus*, Quanjer, 1913; *Potato Leaf-roll Virus*, Appel, 1911; *Potato Virus 1*, J. Johnson's classification.

The Virus

This virus is of historical interest as well as of economic importance ; it was first described and named by Orton in his bulletin of 1914, while Quanjer, in 1916, was the first to demonstrate the infectious nature of the virus. Leaf-roll is of great economic importance and is probably the principal cause of

FIG. 68.

A and B. *Solanum Virus 14* (potato leaf-roll virus).

A. Rolling induced in potato, var. Arran Victory.

B. Rolling induced in potato, var. President.

C. *Solanum Virus 2* (potato virus Y) ; section through a node exhibiting severe necrosis. The whole cortex of the petiole is necrotic and the pith is slightly involved. The main vascular tissues of both stem and petiole are normal. Note normal xylem vessels by which the fallen leaf remains attached to the stem. $\times 75$.

D. *Potato Acronecrosis Disease* (top-necrosis) caused by *Solanum Virus 1* (X virus) and other viruses. Section of stem showing a large necrosis in the vascular tissue. This has spread from a few degenerate phloem groups. The majority of the phloem is normal. $\times 75$.

(C and D, after Bawden.)



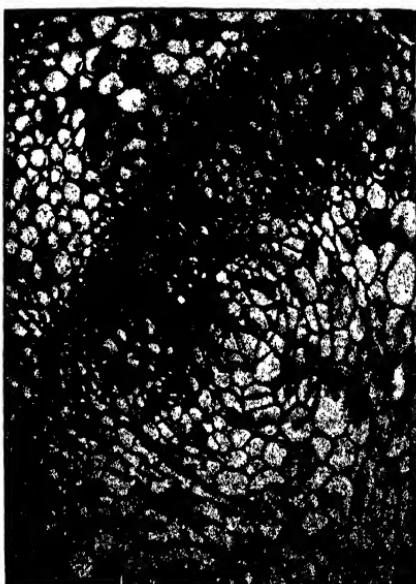
A



C



B



D

potato degeneration. The reduction in yield caused by this virus is considerable. The virus is scarce in the best "seed"-growing districts of Scotland and Ireland, but is comparatively common in the south of England and in some parts of Ireland. In France, in the Bordeaux regions, it is so common as to make it difficult to find a potato plant not infected by it. It also occurs in many parts of North America.

Since the virus is not sap-transmissible, there is no information on its properties.

Transmission. This virus can only be transferred by stem grafting, rarely by tuber core-grafts, and by insect agency. The chief insect vector is the aphis *Myzus persicae* Sulz. (53), and there are two subsidiary aphis vectors, *Myzus pseudosolani* Theob. and *Myzus circumflexus* Buckt. (see Chapter VIII). There appears to be some obligatory connection between the virus and the aphis *Myzus persicae*. This is suggested, firstly, by a delay of forty-eight to fifty-four hours in the development of infective power within the insect, and, secondly, by the fact that the insect retains infective power after long periods of feeding upon non-solanaceous hosts which are immune to the virus (54).

Diseases in Different Potato Varieties caused by *Solanum Virus 14*

President. The first sign of the disease in this variety is a pallor which develops at the edges of the young leaves; later this spreads to the lower leaves, which become thickened and leathery. There is less actual rolling of the leaves in President than in some other varieties, and the whole tendency of an infected President plant is towards a stiff upright habit. Often the young leaves show a pale yellowish coloration on the upper side with development of pink pigment on the lower. The whole plant is very harsh to the touch and rattles when shaken. The leaves ultimately become highly necrotic (see Fig. 68, B). The variety President is extremely susceptible to the virus, and it is largely because of this fact that the variety has fallen out of cultivation.* In many potato varieties growing out-of-doors the disease may be divided into two phases known respectively as "primary" and "secondary" leaf-roll. This distinction is less marked in hot seasons or in plants grown under glass. In the primary condition the rolling and pallor are confined to the young leaves, but in the secondary phase the lower leaflets are also affected, being crisp and dry. Plants infected by means of the aphis late in the season

may show no current season symptoms, but tubers from such plants as a rule give rise to plants which develop the secondary leaf-roll condition only.

The presence of an excess of starch in the rolled leaves is a constant symptom of this disease. The rolling seems to be a direct consequence of the presence in the leaves of this abnormal amount of starch, which causes a distention of the spongy parenchyma (32).

Arran Victory. In the variety Arran Victory diseased plants show a less upright type of rolling than in President, together with a development of purple pigment at the bases of the leaves and the frequent production of small aerial tubers (see Fig. 68, A).

King Edward. The first symptoms commence to appear three or four weeks following infection. A general pallor appears on the young leaves and this is followed by a slight rolling at the base. Pink coloration develops on the lower surface of both young and old leaves. Later fairly pronounced rolling appears on the lower leaves, accompanied by the usual stiffness and leatheryness.

Arran Chief, Kerr's Pink. On these two varieties symptoms develop about four weeks after infection. The first development is an interveinal pallor on the young leaves, this is followed by rolling, accompanied by pink pigmentation, the leaves becoming yellowish on the upper surface, stiff and harsh.

Great Scot. The actual rolling of the leaves in this variety is not pronounced. The main symptom consist of an interveinal pallor accompanied by a stiffness of the lower leaves.

Burbank. In this American potato variety the rolling is particularly prominent in the lower leaves, though it is also evident in the upper ones. The foliage is rigid and leathery, and not soft and flexible as in normal plants. Diseased plants are noticeably stunted and are lighter in colour than are healthy plants.

Tuber Symptoms. In certain potato varieties, particularly in America, the tubers show an internal necrosis plainly visible to the naked eye and known as "netnecrosis." In this symptom the necrosis is restricted to the phloem strands, *i.e.*, the sieve tubes and companion cells. The American variety Green Mountain and the English variety Golden Wonder are examples which show "netnecrosis" of the tubers when infected with *Solanum Virus 14* (leaf-roll virus). Another tuber symptom is the production of long etiolated sprouts known as "sprawling sprouts."

Such sprouts, however, are not an invariable accompaniment of infection by this virus.

Histopathology. In the stems and petioles of affected plants the necrotic process consists of a thickening of the walls of the primary phloem groups together with a slight separation of primary cell walls, so giving rise to small intracellular spaces. Occasionally this is sufficiently severe to cause the obliteration of the sieve tubes. The necroses occur equally in the internal and external phloem, but in the latter they are usually limited to the primary elements. No spread of the necrosis to the neighbouring tissues has been observed (3).

The Physiology of the Leaf-roll Disease. There is apparently no fundamental difference in the nitrogen metabolism of healthy and leaf-roll plants, and the formation of nitrogenous compounds proceeds along the same lines in each case. In diseased plants, when in the "secondary" condition, the photosynthesis is much reduced in the early part of the growing season. The main reactions proceeding in the laminae are conversion of starch to hexose, hexose to sucrose and sucrose back to starch. It has been shown that in healthy plants sucrose is the sugar of translocation, but in leaf-roll plants sucrose is absent in the petioles at any time of the day or night, and sucrose, therefore, plays no part in translocation of leaf-roll material (2). Except for a short period covering the end of dormancy of the tuber to the first unfolding of the leaves, the infected plant respires at a much higher rate than does the healthy one. The rate of respiration is not directly related to the presence of the virus, but rather to the available amount of respirable substrate. Normally the accumulation of such substances in the leaves of leaf-roll plants occurs at a very early stage of development (60).

Diseases in Other Solanaceous Plants caused by Solanum Virus 14

Lycopersicum esculentum. Tomato. The virus can be transferred from infected potato plants to tomato and *vice versa* by the agency of the aphis *Myzus persicae*. Infected tomato plants show no very distinct symptoms. The leaves are very slightly rolled, but they become rigid and somewhat leathery. The plants do not appear to be stunted.

Datura Stramonium, D. tatula. On these two species the disease, when transmitted by *Myzus persicae*, is characterised by

a marked chlorosis and slight rolling of the leaves. As the disease progresses the leaves become leathery in texture.

Solanum dulcamara. Woody or bitter nightshade. On this species there may be two types of reaction; in one the disease is severe and the plant ceases to grow, becoming stunted with the characteristic stiff leathery leaves. In the other type of reaction the plant carries the virus without the production of symptoms.

Solanum villosum. This weed is readily infected with the virus by means of the aphis. The symptoms are of the usual type, chlorosis, rolling and leatheriness of the leaves (19).

SOLANUM VIRUS 15. Hungerford and Dana

Synonyms. *Potato Witch's Broom Virus*, Hungerford and Dana, 1924; *Potato Wilding or Semi-wilding Virus*, Anon., 1927; *Tomato Witch's Broom Virus*, Young and Morris, 1928; *Tobacco Witch's Broom Virus*, Young, 1929; *Potato Virus 11*, J. Johnson's classification.

The Virus and its Transmission. The virus appears to be widely distributed in North America and occurs in all areas of Oregon. It is considered by Murphy to be the same virus as that causing the disease known as "wilding" or "semi-wilding," which occurs in the British Isles. The disease does not seem to be of very great economic importance.

There is no information available on the properties of this virus, as it is not sap-inoculable. It has only been transmitted experimentally by means of top-grafts and by core-grafting the tubers. It is not known how the spread is achieved in the field, but various species of aphides have failed to transmit the virus under experimental conditions.

Diseases in Different Potato Varieties caused by *Solanum Virus 15*

The primary and secondary symptoms of the witch's broom disease are usually distinct, although they may overlap. The first symptom is an increasingly prominent flavescence, a type of chlorosis, in the new leaflets on one or more stems. Marginal flavescence consisting of light green or yellow margins on green leaves is a common symptom.

Primary Symptoms. The tops of potatoes showing the

first symptoms of witch's broom grow rapidly, producing new leaflets that are dwarfed, flavescent and often rugose. The stems bearing them are also flavescent and have unusually long internodes and enlarged nodes. Such stems are cylindrical rather than quadrangular. The lower leaves are normal in appearance on potatoes with primary witch's broom because these will have developed before the onset of the disease. In the field the tops of affected potato plants often become purple and the flavescent margins of the leaflets frequently become pink or red. Many flavescent, spindling axillary branches develop all along the stems and bear tops with typical symptoms. Basal branches also may arise and the plant soon assumes a bushy appearance. The tops of the plants often bloom and produce fruit in abnormal profusion. Late in the growing season the subterranean tubers sprout and send up very many spindling little stems with dwarfed leaves around the base of the main stem. These tubers sometimes proliferate and produce chains of a few small tubers. Many little aerial tubers with leafy eyes commonly develop on the main stems. Plants showing primary symptoms usually bear 25 to 200 very small subterranean tubers.

Secondary Symptoms. In the further development of a witch's broom plant the basal sprouts grow slowly while the main stems grow rapidly. The old leaves produced while the plant was normal gradually die and are replaced by the dwarfed, chlorotic, often simple leaves produced by the spindling tops and branches. Necrosis of the phloem in the bases of the stems of American potato plants is also a feature of the disease. The main stems finally die, completing the transformation of a large normal potato plant into a dwarfed plant with secondary symptoms. While large plants with witch's broom usually bear numerous tubers, plants extremely dwarfed by the disease often produce very few. Pubescent, usually leafless, filamentous branches, $\frac{1}{4}$ to 2 mm. in diameter and $\frac{1}{2}$ to 10 cm. long, often appear at many of the nodes of the stem. While this symptom is frequently absent on infected plants, especially in the field, it is very useful in diagnosis when it occurs. These slender, even thread-like, aerial stems are often branched and bear terminal tubers and simple leaves (68). On the English potato variety President the virus produces a similar symptom picture, an outstanding feature being the extraordinary number of small tubers formed, one of the President plants in the second season producing 116 (86).

Tuber Symptoms. Sprouts from infected tubers show

symptoms of the disease soon after they appear, for each tuber may send out any number up to fifty of spindling little stems with dwarfed and often simple leaves. These spindling sprouts are a characteristic symptom of the disease (see Fig. 66, C). A common feature in diseased President and Arran Victory varieties is the premature sprouting of the tubers during the autumn or even in the drill before digging, with the production from every eye of spindling shoots, which send out further lateral shoots from their basal portions. Netnecrosis of the tubers is also a symptom of the witch's broom disease.

Diseases in Other Solanaceous Plants caused by *Solanum Virus 15*

Lycopersicum esculentum. Tomato. The first symptom is marginal chlorosis or flavescence in the new leaflets, which is a characteristic symptom of the witch's broom disease. The terminal leaflets then turn light yellow, pale green, hyaline or purple. The leaflets are much dwarfed and often rugose, while many of them have very narrow leaf blades or none at all. The leaflets and rhachises are prominently downwards curled and not rolled upwards. Affected plants frequently produce many small insipid tomatoes containing few seeds. Only a small percentage of these seeds are of normal size and viability. Senescence is not much delayed in tomato plants with witch's broom. The normal old leaves die off, leaving the flavescent, curled, nearly bladeless leaves on the stems. The mature stems of affected tomato plants turn yellow and become hollow, tough, and partly dry.

Histopathology. Examination of the flavescent or chlorotic leaves of diseased tomato plants shows the palisade cells to be abnormally short with very little chlorophyll. The margins of the leaves are much thinner than normal.

Nicotiana tabacum. Tobacco, var. White Flowering. Tobacco plants of the above variety when infected by grafting with the witch's broom disease show the following symptoms. Numerous slender axillary branches develop, bearing greatly dwarfed leaves, while new leaves developing on the main stem after infection are abnormally small. Many of these leaves show marginal flavescence and most of them are entirely flavescent. Flowers are present, but they are only half as long and thick as normal flowers of this variety. Later, rosettes of little leaves only 1 to 2 cm. long appear on the stems. Some of the leaves may turn white with green veins and necrotic spots. The main characteristics of this disease

are the marginal flavescence of the leaves and the abnormally numerous, spindling, axillary and basal branches.

Histopathology. The marginal palisade cells in affected tobacco leaves are unusually broad and have prominent inter-cellular spaces. The edges of the diseased leaves are thinner than those of healthy leaves and contain only about half as many chloroplasts (62).

SOLANUM VIRUS 16. Barrus and Chupp

Synonyms. *Potato Yellow Dwarf Virus*, Barrus and Chupp, 1922; *Potato Virus 5*, J. Johnson's classification.

The Virus and its Transmission. There seems to be no information available on the physical properties of this virus. The virus appears to be transmissible by mechanical methods and the insect vectors are said to be the aphides *Myzus persicae* Sulz. and *Macrosiphum gei* and the potato leafhopper *Empoasca fabae* Harris (see pp. 538, 532) (23A). There seems to be some doubt as to the actual vectors of this virus, since Black states (9) that the clover leafhopper, *Aceratagallia sanguinolenta*, has been found to be a vector of the virus. This insect becomes able to transmit the virus about nine days after feeding on diseased plants and is capable of harbouring the virus from early November until early April. Black further states that he has been unable to confirm that *M. persicae*, *Macrosiphum gei* (*solanifolii*) and *Empoasca fabae* can transmit this virus.

Diseases in Potato caused by Solanum Virus 16

Affected potato plants have a stocky, dwarfed appearance and the stems are yellowish-green in colour. The growing apex dies early. The stems when split show rusty specks in the pith and cortex of the nodes, and sometimes also of the internodes, of the upper portion of the plant. This character is a useful diagnostic symptom. The leaflets are generally rolled, but sometimes corrugated.

Tuber Symptoms. The symptoms in the tuber are characteristic. The tubers are usually small, few in number, set close to the stem and sometimes cracked. The flesh is often discoloured with rusty-brown specks or areas in the pith and other internal tissues of the tuber, but this discolouration is seldom found in the fibro-vascular bundles.

These discoloured areas are most pronounced in the middle or bud end of the potato as taken from the field, the stem end not being affected except during storage. The old seed tuber usually remains unrotted in the ground (1).

Host Range. According to Black (8), red clover, *Trifolium pratense*, is susceptible to the virus.

SOLANUM VIRUS 17. Whipple

Synonyms. *Potato Yellow Top Virus*, Folsom, 1926; *Potato Apical Leaf-roll Virus*.

The Virus and its Transmission. The virus can be transmitted by core-grafts, but there is at present no other information on the transmission or properties of this virus. The disease has been recorded from Montana and Maine in the United States of America.

Diseases caused by Solanum Virus 17

Yellow Top; Apical Leaf-roll. Infected plants are slightly dwarfed and show a rolling of the upper leaves similar to "primary" leaf-roll (*Solanum Virus 14*). Apical leaf-roll can be distinguished, however, from the primary leaf-roll by the fact that it persists in the upper leaves, only, in succeeding generations. The tubers are reduced in size, but normal in shape and number, and are frequently, but not invariably, attached to short stolons resembling the rather clustered tuber sets of plants affected with witch's broom disease (*Solanum Virus 15*). Apical leaf-roll plants are distinguished from those affected with witch's broom by taller, more vigorous and fewer shoots which form larger and fewer tubers.

The following American potato varieties are susceptible to the virus: Green Mountain, Rose No. 4, Irish Cobbler and Bliss Triumph. The first two manifest symptoms earlier in the season than the other two varieties (50).

SOLANUM VIRUS 18. Richards

Synonym. *Psyllid Yellows Disease Virus*, Richards, 1928.

The Virus and its Transmission. Little is known concerning this virus; the insect vector is the potato and tomato psylla, *Paratriozza cockerelli* Sulc (see p. 496). The virus is carried in the



FIG. 69.
A. Acropetal Necrosis (rugose mosaic) on potato Up-to-Date ; the plant contains *Solanum Virus 1* and a virus of the *Solanum Virus 2* type (X plus Y).
B. *Solanum Virus 18* (psyllid yellows virus) on potato Irish Cobbler : note basal rolling and cupping of leaflets.
(A, after Murphy ; B, after Richards and Blood.)

tubers of affected plants. The disease caused by this virus was first described by Richards (43), in 1928, from Utah, U.S.A., and later by Shapovalov (52) in California.

Disease caused by *Solanum Virus 18*

Psyllid Yellows. An upward rolling of the basal portion of young leaves constitutes the first and most distinctive symptom. In the Bliss Triumph and Irish Cobbler varieties of potato, this rolled portion becomes brilliantly coloured, varying from light pinkish-yellow to purple. The older leaves roll upward, turn yellow and die. The axillary buds are stimulated into one of, or a combination of, the following three types of growth : thick shoots which may exceed the leaf in length, aerial tubers, and rosettes of small and frequently highly coloured leaves (see Fig. 69, B).

Geographical Distribution. The psyllid yellows disease has only been observed in the United States of America, where it has been recorded from Utah, Idaho, Montana, Wyoming and California. It has not been observed in Europe.

Description of the Composite Mosaic Diseases of the Potato Crinkle (= American Mild Mosaic). Component Viruses. *Solanum* Viruses 1 and 3 (Potato Viruses X and A)

Symptoms on Different Potato Varieties. The following description of the crinkle disease is given by Murphy (31), and may be taken as fairly typical for this disease on many potato varieties. Affected plants are bushy, dwarfed specimens of about the same size as the low-headed type of leaf-roll plants (*Solanum Virus 14*). The colour is a pale green, but this feature is not marked.

The most characteristic symptom is a pronounced puckering and downward curving of the leaves. There is no distinct spotting as in mosaic (*Solanum Virus 1*), but diffused slightly yellowish areas occur all over the foliage. As death approaches, this colour becomes more pronounced and is accompanied by rusty-brown spots, beginning near the tips of the leaves. The foliage is brittle and easily injured. There does not appear to be in crinkle any discolouration of the vascular tissue of the leaf or stems. Since only one constituent virus of the crinkle disease is aphid-borne, it follows that crinkle is not transferred *as such* from diseased to healthy plants in the field. The aphid-borne virus (*Solanum Virus 3*) is presumably brought independently to plants already

infected with *Solanum Virus 1* (virus X) and those plants then develop the crinkle disease.

Under laboratory conditions the whole disease is transmitted from potato to potato by grafting, but sap-inoculation transmits only one constituent, *i.e.*, *Solanum Virus 1* (virus X). It is possible, however, that improvements in the inoculation technique, such as the addition of carborundum powder to the inoculum, may enable the whole complex to be transmitted in this way.

Crinkle in var. President. Fairly well-defined pale patches form first in the apical leaves of the new growth. Contemporary with the formation of these pale patches there occurs a shrinkage of the tissues involved, and in consequence a varying degree of deformity. Accompanying this mottle and deformity there is a very definite waving of the edges of the leaflets (see Fig. 70, B). Occasionally small black necrotic spots or fine necrotic streaks are present in conjunction with the veins. In the variety Irish Chieftain the symptoms of crinkle are similar (44) (see Frontispiece).

Crinkle in var. Arran Victory. The disease is less severe in this variety than in the foregoing. In Arran Victory the necroses and deformity are rare and never severe, and waving and rugosity of the leaf are less prominent. Mottling may, however, be very distinct, and its formation as chlorotic patches extending from the cleared veins to the angles between them is characteristic. The variety Arran Chief reacts similarly to the double infection (44).

Crinkle in var. Arran Comrade. In this second early variety the reaction is intermediate in severity between that of President and Arran Victory. Clearing of the veins is the first symptom, and this is followed by a diffuse mottling. Necrosis and deformity are unusual, but the waving of the edge of some leaflets may be pronounced.

The typical crinkle symptoms can only develop on varieties which are tolerant of both viruses, hence the disease is *never* seen in Arran Crest, Epicure, British Queen, Up-to-Date, Kerr's Pink, Great Scot, etc. It is for this reason that Murphy has stated recently (33) that *Solanum Virus 3* (virus A) in combination with *Solanum Virus 1* (virus X) determines the survival of British varieties, and the really great varieties, such as Up-to-Date, British Queen and Kerr's Pink, are so intolerant as to throw off the disease. This does not minimise the importance of *Solanum Virus 2* (virus Y); there are no varieties which escape this so well through intolerance, or escape its effects through tolerance, but it is scarce in the best seed potato-producing districts, and the



B

FIG. 70.

A. *Solanum Virus 7* (paracrinkle virus) on potato Arran Victory.
B. *Solanum Viruses 1* and *2* (X and A) on potato President, causing crinkle disease.
(A, after Murphy.)

basis of " changing seed " each year is the avoidance of *Solanum Viruses 2 and 14* (virus Y and leaf-roll virus). On the other hand, *Solanum Virus 3* (virus A) is more common, apparently, in the " seed " potato areas, and it can be controlled partly by changing " seed " and partly through intolerant varieties. The comparatively good, but still short-lived varieties, such as Champion, Arran Chief, Arran Victory and Arran Banner, tolerate *Solanum Viruses 1 and 3* (X and A) and show severe crinkle.

Rugose Mosaic. Component Viruses. *Solanum Viruses 1 and 2* (Viruses X and Y)

Symptoms of Rugose Mosaic on Different Potato Viruses. This disease occurs most commonly in the United States of America, since one of the two constituents, *Solanum Virus 1*, is present in all commercial potato stocks in that country. The chief characteristics of the disease are as follows : in severe cases the plants are dwarfed and the tubers reduced in size. The lower leaves generally have black necrotic veins, while the upper leaves are mottled with light green spots (see Fig. 69, A). This mottling is very pronounced on certain American potatoes, such as Bliss Triumph. The chlorotic spots are relatively small, usually about $\frac{3}{16}$ inch across. They are numerous and commonly most abundant near the leaf-ribs on the younger leaves. The foliage is wrinkled or ruffled, particularly on Bliss Triumph and less so on the Irish Cobbler and Ohio varieties. High temperatures may mask the mottle symptom, but the roughness or rugosity and the dwarfing persist (10). The disease is usually severe in the Green Mountain variety. Affected plants are dwarfed and curled, with rugose, abnormally hairy leaves. Such plants are frequently killed before the production of tubers. The American varieties Early Rose, Early Ohio, Irish Cobbler and Rural New Yorker seem somewhat resistant to infection with rugose mosaic.

In the potato variety Institut de Beauvais symptoms of rugose mosaic can apparently be produced by the action of *Solanum Virus 2* (virus Y) alone (84).

As a conclusion to this section on the composite virus diseases of the potato plant, some remarks from a recent paper by Murphy (88) may be quoted here, as they help to clarify what is admittedly a difficult and confusing situation. It is probable that no virus is entirely harmless, although in some cases no natural spread or adverse effect on yield has been demonstrated. Omitting such, it is found that the principal potato mosaic diseases are

caused by the four viruses, *Solanum Viruses 1, 2, 3, 8* (viruses X, Y, A and F), alone or in combination, and these form the types of three natural groups, *Solanum Viruses 2* and *3* (Y and A), though distinct, being at present considered together on account of the occurrence of intermediate forms. These groups are very shortly characterised as follows :

Group 1. Viruses of the *Solanum Virus 1* type. Mottle (or rings) on *Datura* and tobacco ; pass L_3 filter ; insect vector unknown (not *Myzus persicæ*) ; typical disease on potato, simple mosaic.

Group 2. Viruses of the *Solanum Virus 8* type. Spots on *Solanum nodiflorum* and *Capsicum annuum* ; carried without symptoms by tobacco and *Datura* ; held by L_3 filter ; insect vector *Myzus persicæ* under certain conditions ; typical disease on potato, yellow or aucuba mosaic.

Group 3. Viruses of the *Solanum Virus 2* or *3* type. Vein-banding on tobacco, not inoculable to *Datura* ; held by L_3 filter, insect vector *Myzus persicæ* ; typical disease on potato, veinal mosaic or leaf-drop streak.

The four viruses mentioned, together with their combinations, produce eight diseases as follows : though complexes of more than two also occur.

- (1) *Solanum Virus 1* (virus X) . Simple mosaic.
- (2) *Solanum Virus 8* (virus F) . Yellow mosaic.
- (3) *Solanum Virus 3* (virus A) . Veinal mosaic.
- (4) *Solanum Virus 2* (virus Y) . { Veinal mosaic.
Leaf-drop streak.
- (5) *Solanum Virus 1* plus
Solanum Virus 8 . . . Interveinal mosaic.
- (6) *Solanum Virus 1* plus
. *Solanum Virus 3* . . . Crinkle.
- (7) *Solanum Virus 1* plus
Solanum Virus 2 . . . { Rugose mosaic.
Leaf-drop streak.
- (8) *Solanum Virus 3* plus
Solanum Virus 8 . . . Double virus aucuba mosaic.

The first three diseases are normally passed over in the field, the eighth is not very common, and the remainder make up the "mosaic" of the practical man. These, as will be seen, are caused by a virus transmitted by *Myzus persicæ*, occurring alone or in combination with *Solanum Virus 1*. This, therefore, reduces the mosaic problem to two dimensions : the control of *Myzus persicæ*,

which is only the leaf-roll problem over again with the additional complication of carriers, and the control of *Solanum Virus* 1. The latter is still an enigma, for though it spreads so freely as to be almost ubiquitous, occurring, for example, throughout the variety Up-to-Date, and in all American commercial potatoes without exception, its vector is unknown (unless, as the writer has suggested, the vector is a flower-feeding species of thrips). The virus infiltrates rapidly into virus-free stocks even when grown in isolation in the best districts, and is not entirely unknown in experimental glasshouses. As part of the basis of all the severe mosaic it is an active danger, but the question still awaits answer whether it is economically possible or necessary to control it as well as the aphid-borne viruses.

Geographical Distribution of Potato Viruses. While a certain number of the viruses affecting the potato plant are widely distributed throughout the new and the old worlds, there are others which are found only in the former. The following viruses occur in Europe and North America, and are probably found wherever the potato is grown. *Solanum Viruses* 1, 2, 3, 4, 6, 9, 14 and 15. Certain potato viruses, however, appear to be found only in North America ; these are *Solanum Viruses* 11, 12, 13, 16 and 17. On the other hand, one or two seem to be confined to Europe, such as *Solanum Viruses* 5, 7 and 8.

The practical absence from north-western Europe of many serious American afflictions must be attributable to failure to maintain the requisite balance between virus and potato, as a result of which they die out, for it is practically certain that most of them must have been introduced in tubers at some time, as, indeed, remnants of such diseases as witch's broom (*Solanum Virus* 15) show.

Control. The magnitude of the question of the control of potato virus diseases is shown by the fact that in the British Isles alone there are half a million acres of potatoes in England and Wales, and of these some 120,000 acres are planted with Scottish or Irish seed potatoes annually. The balance is planted with local "seed," and it is a question to what extent this could be profitably replaced, for fresh seed is estimated to increase yields by one ton per acre.

The question of the control of potato virus diseases is best approached from two main standpoints. The first of these is the propagation of virus-free "seed" potatoes under conditions where the risk of spread of infection is reduced to a minimum, and the

second is the production of virus-resistant or tolerant potato varieties. In producing a nucleus of virus-free stocks the method of "indexing" is practised. This consists in testing the health of the selected tuber stocks by growing a portion of each tuber during the winter under forcing conditions in the glasshouse. A convenient method of obtaining a representative portion of a tuber is to extract, by means of a sterile cork borer, a plug of tissue bearing an eye. This method of tuber-indexing is used to eliminate the virus-infected tubers and to obtain healthy foundation stocks. The stocks are next multiplied in isolation under conditions where contamination is unlikely. It is important to include in the indexing method described above inoculation tests to differential hosts such as tobacco and *Datura* as well as examination of the external appearance of the plants. By so doing potato plants which are symptomless carriers of viruses will also be eliminated. It has been shown that localities suitable for the multiplication of virus-free potato stocks are at high altitudes, on the sea coast or in clearings in the midst of softwood forests where the aphid vectors are scarce or absent. When it can be avoided, potatoes for "seed" production should not be grown in the vicinity of *Brassica* crops, since the aphides hibernate on these plants.

As regards the production of virus-resistant varieties of potatoes, much remains to be done in this direction. Although it may be difficult if not impossible to develop a potato that is resistant to all potato viruses, yet results of some recent work show genetic segregation for resistance to certain of the mosaic-type viruses, and it may eventually prove possible to produce varieties resistant to more than one virus. Further, as Schultz has pointed out (49), potato localities vary regarding the presence and spread of virus diseases; it is uncommon to find all the reported viruses in a single potato-growing area, usually only a few of them requiring serious attention. Also the insect vectors of viruses vary from one locality to another and from season to season in the same locality.

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CHAPTER VII

Ananas Virus 1 ; **Musa Viruses 1-3** ; **Tulipa Virus 1** ; **Lilium Virus 1** ; **Allium Virus 1** ; **Iris Virus 1** ; **Freesia Virus 1** ; **Saccharum Viruses 1 and 1A-1G and 2-5** ; **Zea Viruses 1-3** ; **Triticum Viruses 1 and 1A** ; **Oryza Viruses 1-2**.

ANANAS VIRUS 1. Linford

Synonyms. *Pineapple Yellow-spot Virus*, Illingworth, 1931 ; *Pineapple Side Rot (Virus)*, Sideris, 1927.

The Virus and its Transmission. Little is known of the virus which causes the yellow-spot disease of pineapples. It does not appear to be sap-inoculable from the pineapple, but it may be so from other host plants. The insect vector is the onion thrips, *Thrips tabaci* Lind. (see p. 462). Larvae from a non-infective colony of thrips become infective after feeding upon a diseased plant, but adult thrips treated similarly do not. The virus survives in the insect during pupation, and insects that feed on a source of virus, while larvae may be infective as adults. There is a period of approximately ten days after first feeding on a diseased plant before the thrips can transmit infection. A single insect, larva or adult is capable of transmitting the virus (28). The possibility must be borne in mind that this virus and *Lycopersicum Virus 3* (tomato spotted wilt virus) may be the same, although this is by no means proved. The symptoms caused by the two viruses on certain hosts, however, are very similar, and the same insect transmits both viruses.

Differential Host

Emilia sagittata (Vahl) D.C. This weed is very susceptible to infection with *Ananas Virus 1*, and is frequently found infected with the virus in the vicinity of diseased pineapples. Symptoms consist of a marked mosaic mottling, frequently of a zonate or ringed character. A mottling in the form of dark and light green stripes develops on the involucres of the flower heads immediately following infection. The production of mild circular blotching or patterns of concentrically zonate lines of dark and light green is



FIG. 71. *Ananas Virus 1* (pineapple yellow spot virus).

- Six months' old pineapple, var. Smooth Cayenne; note stunting and dwarfing of the central leaves in contrast with the lower apparently normal leaves.
- Longitudinal section of young pineapple showing browned patches in the stem around the attachment of the leaf which had the "initial spot."
(After Serrano.)

extremely reminiscent of the behaviour of *Lycopersicum Virus 3* (tomato spotted wilt virus).

Diseases caused by Ananas Virus 1

Bromeliaceæ

Ananas cosmostus. The pineapple. *Yellow-spot disease* (see Fig. 71). The disease is manifested first by a slightly raised circular yellowish spot, the so-called "initial spot," on the upper surface of a young tender leaf. It varies in size, ranging from 2 to 15 mm. in diameter. The evidence suggests that infection takes place somewhere near the base of a very tender leaf, but owing to the continued basal growth its first appearance is usually observed at about 8 cm. from the base of the leaf in the third or fourth whorl. Ordinarily only one leaf shows the initial spot, although two or more have been observed with the same infection in some instances. Below the initial spot a yellow streak develops after a few days, extending down the base in the form of a water-soaked spot, and under favourable conditions rot soon follows. In a few days more the yellow streak may show up, extending to the leaf immediately above the first affected leaf, and thence to all the central leaves. These leaves become stunted, chlorotic, and somewhat brittle and tend to tighten together, while all the lower leaves remain apparently normal. When cut lengthwise, diseased plants reveal patches of browned tissue in the stem around the attachment of the leaf which had the initial spot. At this point the plant evidently ceases to grow and shows a tendency to bend downwards, owing to the force of the growth of the opposite side.

Infection may take place at any time during the life of the plant, although it has been observed more frequently among younger plants. Early infections prevent normal development of the plant, causing stunting, dwarfing and ultimate death long before maturity. Late infections are usually confined to fruits and crowns. The fruits can be infected through one or more fruitlets of very tender age. This may happen during the blooming period. Infection sometimes starts from a flower or a young fruitlet, in the same manner as in a young leaf, or the crown of a young fruit. Malformation, dwarfing, yellowing, and rotting of a few "eyes" of one side of the fruit result from this type of infection, while the opposite side develops normally, forcing the fruit to bend towards the focus of infection. In cases of advanced infection the fruit may become hollow (46).

Leguminosæ

Pisum sativum. Garden pea. *Streak*. A streak disease of the garden pea is produced by infection from yellow-spot pineapple (27). A streaking and spotted brown necrosis of the pods, stems and leaves develops. The pods show necrotic circular pitting or they may collapse altogether; on the leaves the injury may begin with spotting or brown vein-streaking, extending down the stems and associated with phloem necrosis (*cf.* this disease with streak of peas produced by *Lycopersicum Virus 3*, tomato spotted wilt virus, p. 300).

Host Range. The host range of *Ananas Virus 1* appears to be very wide, and in this respect also it resembles *Lycopersicum Virus 3* (tomato spotted wilt virus). According to Linford (28) the incomplete list of hosts includes members of the following families: Bromeliaceæ, Liliaceæ, Caryophyllaceæ, Leguminosæ, Labiatæ, Solanaceæ, Rubiaceæ and Compositæ.

Geographical Distribution. Yellow-spot of pineapples is one of the major diseases of that plant in the Hawaiian Islands, where it was first reported in 1931. The disease is also present in the Philippine Islands, its occurrence there being first noticed in 1928.

Control. The control of pineapple yellow-spot resolves itself into three main problems, the destruction of the thrips, the eradication so far as practicable of virus-harbouring weeds like *Emilia* spp., and the production of resistant varieties of pineapples. At present the first method is the most promising. Tobacco dust (1·2 per cent) has proved superior to other forms of nicotine for the destruction of the thrips. The incidence of the disease has been found to be correlated with the growth and succulence of the plants, yellow-spot being less prevalent in the poorly growing areas of the field. The relatively slight incidence of the disease in the tobacco-dusted plots is thought to be due to the retardation of growth and toughening of the tissues resulting from the application, rather than to any direct insecticidal effect on the vector, *Thrips tabaci* (6).

MUSA VIRUS 1. Magee

Synonyms. *Banana Bunchy Top Virus*; *Banana Virus 1*, J. Johnson's classification.

The Virus and its Transmission. There is no information on the physical or other properties of this virus, since it has only recently been shown to be sap-transmissible (Soliman *in litt.*).

The specific insect vector is the dark banana aphis, *Pentalonia nigronervosa* (see p. 543).

Disease caused by Musa Virus 1

Musaceæ

Musa cavendishii. Cavendish banana. *Bunchy top*. A plant may show symptoms of bunchy top at any stage of growth, from its first emergence from the ground or as a plant fully grown and throwing a bunch. The name aptly describes the disease; the leaves of a badly infected plant are bunched together at the apex of the plant to form a rosette. Owing to the failure of the leaf stalks to elongate, the leaves stand more erect than is normal. Infected plants are markedly stunted, there being little increase in height once the plant has taken the infection.

The first external symptoms of bunchy top appear in the leaves of the plant. The normal leaf emerges from the centre of the pseudo-stem with the leaf blade wrapped tightly round the mid-rib in the form of a rod or "pipe." The leaf remains tightly rolled until it is almost fully emerged, and then commences to unfurl more or less evenly along its whole length. While unfurling the leaf stands erect, and when fully unrolled the elongation of the leaf stalk carries the blade clear of the pseudo-stem. In the case of secondary infection, it is in a leaf which has unfurled in this manner that the first symptom of bunchy top is usually observed. The first definite symptom of the disease is the appearance of irregular, nodular, dark green streaks about 0.75 mm. wide along the secondary veins on the underside of the lower portion of the leaf blade, along the leaf stalk, or along the lower portion of the mid-rib. In the first instance one, two or several of these streaks may be present. Usually others appear later in the same region. In character they may vary from a series of small dark green dots to a continuous dark green line with a ragged edge, an inch or more in length. When, as is usually the case, the first symptoms take the form of a few characteristic green streaks in the lamina, mid-rib or petiole, this "first-symptom" leaf is otherwise normal. In the following leaf, however, while the "pipe" is still unfurled, pale whitish streaks are seen along the secondary veins of the leaf blade. The leaf also unfurls in a slightly abnormal manner, beginning to unroll from the top and presenting a funnel-like appearance. The presence of the characteristic broken dark green streaks along the secondary veins of the lamina & along the mid-

rib or petiole is the most definite and reliable symptom of bunchy top. These streaks appear as the earliest external indication of the disease. The dark green streaks are not apparent when viewing the dorsal surface of the leaf in reflected light. The leaf should be inspected from the underside so as to allow light to pass through it. Succeeding leaves are more abnormal in appearance, and after several have appeared extreme congestion is apparent at the apex of the pseudo-stem, giving rise to a "rosetted" condition.

In colour the mature leaf of a banana plant in an advanced condition of bunchy top is slightly more yellow than the normal, but in a recently infected plant the leaves may be more green than the normal owing to the dark green streaking. Affected leaves are also more brittle and harsh than the healthy leaf, and there is a distinct rigidity and apparent resistance to wilting.

The root system of affected banana plants shows a greater degree of decay than that of a healthy plant.

Histopathology. In the bunchy top diseased plant disorganisation has taken place in the phloem regions of the vascular system. A study of the internal pathological condition of the vascular system of a diseased plant is best made by cutting transverse and longitudinal sections of the mid-rib and leaf stalk of a plant in the early stage of infection.

A comparison of healthy and diseased bundles reveals the following alterations :

(1) In the phloem region there is a suppression of development of the fibrous sheath. The region in which fibres are normally present is occupied by cells which are heavily laden with chromatophores.

(2) The fundamental tissue in the neighbourhood of the phloem has become gorged with chromatophores. In the normal plant this region is almost entirely devoid of chlorophyll.

(3) "Appositional" growth has taken place in the fundamental cells in contact with the phloem. The original cells have been divided up into angular cells by the growth of thin cellulose walls. Growth of these cell walls has apparently taken place in all planes. The newly formed cells contain numerous chromatophores and distinct nuclei.

(4) "Appositional" growth of cell walls along all planes has taken place in the sieve tubes and companion cells of the phloem, giving rise to numerous angular cells along their length. These cells are uninucleate and their nuclei are of an abnormal type. The abundance of these nuclei in the phloem of a primary affected

plant is a most noticeable feature in any transverse or longitudinal section (30).

Host Range. All varieties of banana grown in Australia are susceptible to *Musa Virus 1*. The chief and only commercial variety of banana grown in north-eastern New South Wales and southern Queensland is the Cavendish (*Musa cavendishii*). Other varieties susceptible to the virus are Gros Michel (*M. sapientum*), the Lady's Finger and the Sugar Variety. In Fiji, the Cavendish, Gros Michel, Vi-ma-ma and the Sawaga (*Musa Banksii*) are reported as susceptible.

Manila hemp (*M. textilis*) is reported in Australia and Ceylon as being susceptible to the virus causing banana bunchy top (*Musa Virus 1*), but the bunchy top of Manila hemp or abacá in the Philippines is due to a different virus (see *Musa Virus 2*). The virus is not apparently transmissible outside the genus *Musa*.

Geographical Distribution. Bunchy top has a comparatively wide distribution among banana-growing countries. In Australia the disease is prevalent in north eastern New South Wales and south-eastern Queensland, and is present also in isolated centres of the banana areas of north Queensland.

It has also been recorded from Fiji, Egypt, Ceylon, Bonin Islands and Ellice Islands.

Control. Magee (30) recommends two main methods for the control of banana bunchy top, *i.e.*, exclusion of diseased suckers and plants from a given area and the eradication, or roguing, of diseased plants. As regards exclusion of the disease, this is particularly important where large areas, at the moment free of infection, are concerned. Such exclusion would need legislation to prohibit the transportation of suckers or vegetative parts of *Musa* spp., the registration of all banana plantations, and the destruction of all banana plants in gardens or similar unregistered places. The disease should also be made notifiable in all areas not so far known to be affected with bunchy top.

Eradication of all affected stools, on a most thorough basis, is perhaps the best means of control. Such eradication must be extremely thorough and each diseased plant should be carefully dug out and destroyed, preferably by burning. The following procedure is recommended, to prevent aphides leaving the diseased plant for a healthy one. First pour not less than $\frac{1}{2}$ pint of pure kerosene into the central leaf of the affected plant and other plants connected with it in the same stool. Wait for a few hours to allow the kerosene to trickle round the leaf base, and so kill all

aphides present. Then dig out the stool, including the infected plant and all others connected with it, and chop into small pieces to facilitate drying.

It is very important that all suckers used for planting are certified free of bunchy top infection.

In Queensland the agents of the Banana Industry Protection Board are in a position to advise growers where suitable planting material may be obtained. They should also be consulted regarding the current planting policy, as a planting permit may have to be refused if the spread of the disease is involved (47).

MUSA VIRUS 2. Ocfemia

Synonym. *Abacá (Manila Hemp) Bunchy Top Virus.*

The Virus and its Transmission. There has been some question in the past as to whether this virus was not the same as that causing bunchy top of bananas, but in view of the failure to infect bananas experimentally with the virus from manila hemp, the two viruses are treated here as separate entities, but the possibility must still be considered that the two viruses are closely related (42).

Musa Virus 2 is not transmissible by mechanical methods of inoculation, and there is in consequence no information on its properties. The insect vector is the same as for the preceding virus, *i.e.*, the aphid *Pentalonia nigronervosa* Coq. (see p. 543). The shortest time required by adult aphides to obtain the infective principle is twelve hours. In addition to this time, between twenty-four and forty-eight hours must elapse before the insects are capable of causing infection on healthy seedlings, that is to say, there is a delay in the development of infective power within the insect of twenty-four to forty-eight hours. The incubation period of the disease in fast-growing seedlings is shorter than in slow-growing seedlings; thirty to thirty-two days as compared to sixty to seventy-two days.

Disease caused by Musa Virus 2

Musaceæ

Musa textilis Néc. *Abacá or manila hemp. Bunchy top.* The general symptoms of bunchy top in abacá include reduction in leaf size and narrowing of the blade, sometimes down to a few centimetres from the mid-rib; irregular and more rapid unfurling of young rolled leaves; darkening of the green colour in the leaf sheaths comprising the trunk; shortening of the leaf sheaths so

as to produce congestion and rosetting at the crown of the plant and degeneration of the root system (62).

Ocfemia (40) describes the leaf symptoms as follows : delicate, thin and transparent, membrane-like areas of varying shapes and sizes may develop on the thin chlorotic portions of the youngest leaf either before it unfurls or immediately after. In transmitted light the main veins of leaves showing the symptoms may be seen as transparent lines. These transparent streaks are continuous on the main veins from the mid-rib to the margin and about $\frac{1}{2}$ mm. in width, but broken into 1 to 5-mm. dashes on the secondary veins. On both surfaces of the leaves these dashes appear yellow. If the leaf is furled the streaks sometimes appear water-soaked. The transparency of the primary and sometimes secondary veins is always to be noticed in aphis transmission of abacá bunchy top. Dark green streaks, varying from mere dots to lines several millimetres long, or starting from the mid-rib and disappearing as they reach the yellowed borders, are of great diagnostic value in some varieties of abacá. The dark green lines are about $\frac{1}{2}$ mm. wide and are occasionally present on the mid-rib, petiole and leaf blades.

Host Range. *Musa Virus 2* appears to be confined to *Musa textilis* and its different varieties and is not transmissible to the banana.

Geographical Distribution. The only record of bunchy top of abacá seems to be from the Philippine Islands, where the disease is serious and widespread.

Control. The methods recommended for the control of banana bunchy top are also applicable in the case of bunchy top of abacá. Ocfemia considers that if infested land is rested for nine to twelve months, if only suckers known to be virus-free are used and if young plantations are regularly inspected, it should be possible to rehabilitate the abacá industry in areas where the disease is serious.

MUSA VIRUS 3. Magee

Synonyms. *Banana Mosaic Virus*, Ogilvie, 1927, and Magee, 1930; *Banana Heart-rot and Infectious Chlorosis Virus*, Magee, 1930, and Wardlaw, 1935; *Banana Virus 2*, J. Johnson's classification.

The Virus and its Transmission. Very little is known of this virus and its properties ; it is thought by Magée (31) to be

transmitted by the dark banana aphid, *Pentalonia nigronervosa* Coq. (see p. 548).

Disease caused by Musa Virus 3

Musaceæ

Musa cavendishii. Banana. *Banana mosaic*. The disease takes the form of a severe yellowing and mottling of the younger foliage, followed by a rotting of the heart leaf and central portion of the pseudo-stem. The heart-rot stage of the disease apparently occurs only during the winter months, but the yellowing and mottling may persist throughout the year. In young plants the disease first appears as whitish or yellowish-white streaks, which may extend from the mid-rib to the margin of the most central leaves. These chlorotic areas may vary from narrow streaks to bands $\frac{1}{2}$ inch or more in width. In some leaves the streaks are not continuous, giving rise to a mottled appearance of yellow and green areas. During the summer, infected plants continue to produce this mottled type of foliage, but with the advent of cooler weather many of the plants commence to rot at the heart leaf, the rotting extending down into the corms and finally killing the plants. This disease has so far only been recorded from New South Wales (31), though Wardlaw (62) mentions an apparent mosaic disease of a similar type as occurring in Brazil.

TULIPA VIRUS 1. Cayley

Synonyms. *Tulip "Break" Virus*; *Tulip Mosaic Virus*.

The Virus and its Transmission. It is probable that more than one virus or virus strain are associated with the disease in tulips known as "breaking," but they are dealt with here as one entity pending the discovery of fresh facts. McWhorter (37) states that he has isolated two viruses from "broken" tulips; these he designates as "colour-adding" and "colour-removing" viruses, the latter being dominant. They probably correspond to the "full-break" and "self-break" viruses of McKenny Hughes (21). The virus or viruses causing tulip break can be transmitted by needle-inoculation and by grafting and plugging bulbs with tissue from "broken" bulbs (7). There is no evidence that the viruses are seed transmitted. The insect vectors, in England, are the aphides *Myzus persicae* Sulz. and *Macrosiphum gei* Koch (*Illinoia Solanifoli* Ashm.) in the glasshouse and out of doors, while

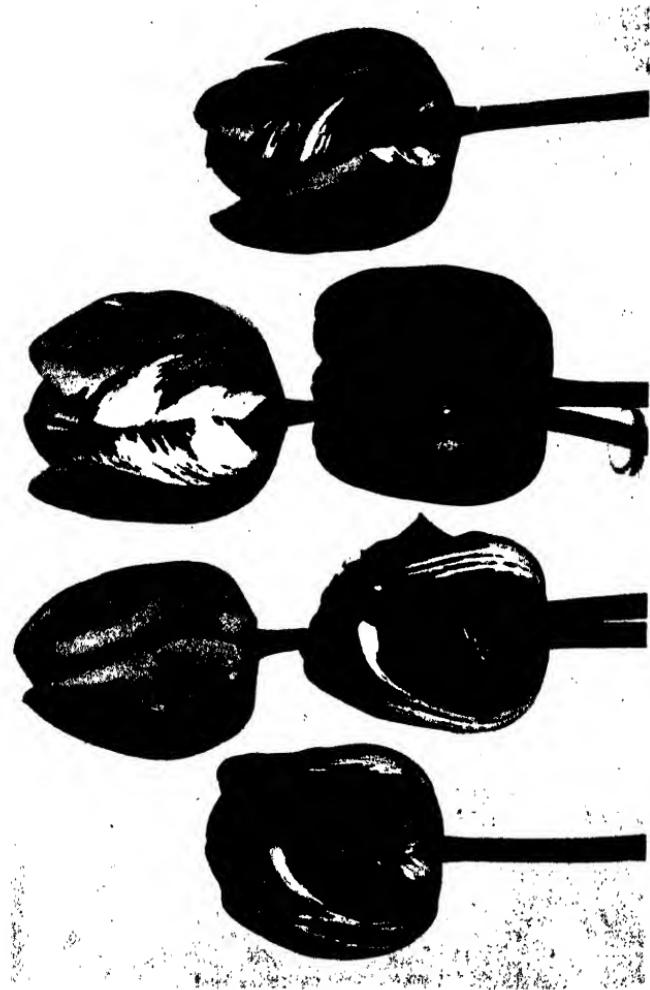


FIG. 72. *Tulipa* Virus 1 (tulip break virus).

Tulip flowers showing the transmission of the virus by reciprocal inoculations between the varieties Farncombe Sanders and Clara Butt. The flower at the left is Farncombe Sanders Rembrandt and that at the right Clara Butt Rembrandt. The upper flowers are Clara Butt control (*left*) and inoculate (*right*) with aphides transferred from Farncombe Sanders Rembrandt. The lower flowers are Farncombe Sanders inoculated by leaf mutation from Clara Butt and control (*right*).

(After McKay and Warner.)

Anuraphis tulipæ B. de Fonsc. is a vector in the bulb store, but not on the growing plant. In Oregon the vectors are the two first named and possibly also *Macrosiphum pelargonii* Kalt. (see pp. 535-537). According to McWhorter (38) it is not possible to separate the colour-adding virus from the colour-removing virus on a basis of their physical properties. The thermal death-point of both lies between 65° and 70° C., they withstand dilutions up to 1 : 100,000, they resist desiccation equally in leaves for eleven days and show the same resistance to alcohol. McWhorter considers that the viruses of tulip break are very similar to that causing mosaic in *Lilium* (see *Cucumis Virus 1*).

Diseases caused by *Tulipa* Virus 1

Liliaceæ

Tulipa spp. Garden tulip. *Tulip break*. The chief symptom of tulip "break" develops in the flower. The change in flower colour is due to a segregation of the anthocyanin pigment in the epidermis of the petal as fine featherings about the margin or in irregular stripes up the middle of each segment, while between the stripes or streaks appear patches of more or less clear ground colour, usually white or yellow. Seedling tulips almost invariably produce self-coloured flowers and are called "breeders" (38). Hughes (21) distinguishes the various types of "break" as follows: "Full break" denotes the type of infection in which the yellow or white mesophyll colour is revealed by the receding "breeder" or "self" colour. "Self-break" is the condition where the "breeder" or "self" colour is intensified into darker streaks or stripes. "Clotting" denotes the type of infection manifested in dark purple or dark red shiny varieties which never show the ordinary bi-coloured "full break," but intensify the self-colour in great splashes and patches (see Fig. 72).

In addition to the flower symptoms, some tulip varieties, but not all, show, when infected, a striping or mottling of the leaves. This mottling is often very indistinct and can only be recognised after a careful examination of the leaves in subdued light. The mottling at first consists of shorter or longer streaks of hardly perceptible silver-grey to light green coloration. Gradually the striping becomes more and more pronounced and gains in depth, especially between the leaf veins, until eventually the greater part of the chlorophyll in the affected part disappears and the leaves become more or less transparent when examined against the light. At this stage the leaves take on a silvery or light greyish-green to

yellowish-green colour, lose their turgidity and become tough (1). In addition the plant is less vigorous, the bulb does not proliferate so freely, and flowering is usually a week or ten days later than in the breeder. The plant, however, does not succumb to the disease; the "broken" variety Zomerschoon, for instance, first described in 1620, is still grown.

Out of eighty tulip varieties inoculated at the Minnesota Agricultural Experiment Station by rubbing the leaves with material from mosaic foliage, thirty-nine (including *Tulipa elegans*) in the Early, Cottage, Darwin, Breeder and Lily Flowering groups contracted the infection.

Tulipa Greigii and *T. Eichleri*. These species are susceptible to artificial infection and show "self" breaking of the flowers. The leaves also exhibit a mosaic mottling.

Histopathology. The cytoplasm, hollowed by numerous small vacuoles, presents a spongy appearance and contains an abundance of lipoids near the nucleus. Normally the epidermal cells of red-flowering varieties are almost entirely filled by a large central vacuole containing a red solution of anthocyanin, while in the mottled areas of diseased flowers some of the small, spherical vacuoles contain this substance and in others the solution is uncoloured (14).

Hyacinthus spp. According to Atanasoff (1), both the hyacinth and the narcissus are susceptible to infection with the tulip break virus (*Tulipa Virus 1*). The different varieties of hyacinth do not show symptoms in the same degree of severity. Slightly affected plants show at first scarcely perceptible light green to light yellow stripes running longitudinally down the veins of the leaves. These stripes are of considerable length, but do not as a rule follow the whole length of the leaf, they often coalesce and form large green or yellow blotches. Gradually the light green or yellow area increases, so that soon the whole leaf and the foliage in general take on a lighter green or yellowish colour. In such cases only a small portion of the leaf area retains, in the form of stripes, the original normal green colour of the leaf, so that the lighter coloured leaves appear as if striped with darker green. The darker green areas are slightly elevated while the lighter green areas are distinctly sunken. The flower stem may also show striping. The striping is usually more pronounced on the upper leaf surface and does not correspond with the striping on the lower leaf surface. Slightly affected plants form quite normal flowers which, however, become smaller and smaller according to

the degree of severity of the disease. Severely affected plants have fewer leaves than normal ; they either form no flower stocks or very abnormal and stunted ones with few flowers. The bulbs of diseased plants are, as a rule, much smaller than those of healthy plants of the same age and variety.

Narcissus spp. *Narcissus stripe* (see Fig. 74, B and C). The so-called stripe disease of narcissus is a widespread and serious disease, and Atanasoff (1) considers that it is caused by the same virus as that which causes tulip break (*Tulipa Virus* 1). For the present, therefore, narcissus stripe is included under diseases caused by that virus. The symptoms on newly affected plants, and especially in the case of the broad-leaved varieties, are at first not pronounced and consist of stripes of a slightly lighter colour than that of the normal tissues. In most varieties the upper side of the leaves shows more mottling than the lower one. As the disease advances the stripes take on a much lighter green to yellow colour. The stripes frequently run the whole length of the leaf and may be quite numerous ; occasionally the flower stock is also striped. Affected plants when viewed from a distance present a greyish-green or silvery appearance, and this has given rise to the American name of "grey disease."

Hodson (*in litt.*) differentiates three kinds of narcissus stripe, classified according to the variety. (1) *Watkin Stripe*, a greater or lesser amount of mottling present, no distortion of the leaf ; there is some evidence in this case that bulbs tend to become reduced in size and vigour over a number of years. Many English commercial varieties are infected, notably Sir Watkin, Golden Spur and Lucifer. (2) *Talma Stripe*, similar to the preceding, but there is in addition definite corrugation and roughening of the leaf surface. It is almost impossible to find a stock of the variety Minister Talma free from the trouble. It is also common in Czarina. (3) *Alfred Stripe*, this is the most virulent of the three, the chlorotic areas are bright yellow in colour, the foliage is severely distorted, and infected bulbs have no commercial value. It is common in King Alfred and Victoria and certain other varieties. All types persist in infected bulbs from year to year and remain reasonably constant for the individual. Symptoms in the flower consist of light or greenish streaking.

Host Range. In addition to *Tulipa* spp., *Hyacinthus* and *Narcissus*, it is possible that some species of *Lilium* are also susceptible to *Tulipa Virus* 1. McWhorter (88) states that the

virus causing "breaking" in tulips is transmissible to lilies, in which it produces a disease indistinguishable from lily mosaic (see *Cucumis Virus 1*).

Geographical Distribution. *Tulipa Virus 1* is very widely distributed and probably occurs wherever the tulip is grown. It has been recorded from the British Isles, Holland, France, Bulgaria, and the United States of America. The literature gives proof that "breaking" became common as soon as tulips were grown in Western Europe, and there are indications that it already existed in Turkey before this.

Control. Control methods consist of careful roguing of infected bulbs, and the regular fumigation of glasshouses and of stored bulbs. If a valuable new tulip seedling "breaks," instead of discarding the whole plant, it is worth while saving the remoter laterals, as they may have escaped infection, and if grown on separately under aphis-free conditions may remain "unbroken." On the other hand, however, since the virus can reach the buds which form, the same season, inside the new main bulb at the base of the freshly "broken" flowering shoot, the laterals from that bulb will show "breaking" in subsequent seasons (7). There is evidence which suggests that the plant becomes immune from virus infection at a certain stage. Presumably this is correlated with the time at which the new bulbs and offsets become cut off by dead tissue from the still growing and flowering stem which perishes with the season of flowering. In the garden, instances are occasionally seen of what was planted as a single breeder bulb producing its main flower "broken," but also an offset with a breeder flower, or *vice versa*. It was formerly supposed that the early tulips, varieties or species, did not "break," whereas after experimental infection they prove liable to "break" just as easily as the May-flowering garden tulips. It may be concluded that these early tulips have passed the stage of susceptibility before the aphis normally reaches the garden (21).

LILIMUM VIRUS 1. Ogilvie

Synonyms. *Lily Yellow-flat Virus*, Ogilvie, 1928; *Lily Rosette Virus*, Ogilvie.

The Virus and its Transmission. The virus is not sap-inoculable and there is no evidence of seed or soil transmission. There is no information on the properties of this virus.

The insect vector has been shown by Ogilvie (43) to be *Aphis gossypii* Glover (see p. 515).

Diseases caused by *Lilium* Virus 1

Liliaceæ

Lilium longiflorum, var. *eximium*. Easter lily. *Lily rosette*.

The disease caused by this virus in the Easter lily was called "yellow flat" by the grower who first observed it, later the name was changed to "lily rosette," as being more descriptive of the symptoms. In plants which have arisen from infected bulbs, the leaves, especially the upper and youngest leaves, curl downwards in a marked manner. They are not appreciably shorter than the normal leaves, but appear so on account of the downward curling. In some cases the leaves twist sideways and may be somewhat distorted. They do not form a shallow trough as in normal plants, but have their upper surfaces flat or rather convex in cross-section. The colour of diseased leaves is slightly chlorotic, but streaks or spots are absent. The general appearance of the plant is a flat rosette or cylinder in contrast to the pyramidal shape of a healthy plant. Current-season symptoms are very similar. Leaves already mature at the time of infection are not visibly affected, but the fresh growth shows the characteristic symptoms. In the case of plants infected late in the season, the leaves are prone to be twisted from side to side, and the internodes tend to be longer than in the case of typically diseased plants. This effect may be due to high temperatures. The topmost leaves are often extremely twisted. The effect of the virus on the size and shape of the bulbs is very marked. The outer scales remain normal and loose; the inner, produced subsequent to infection, are tightly drawn together. The circumference of the bulb is smaller than that of a normal bulb. Another noticeable effect is a tendency towards splitting of the bulbs. The result of this is a continued reduction in the size of the bulbs from year to year until they are little larger than peas (48) (see Fig. 78, B).

The virus has also been observed affecting *Lilium longiflorum*, var. *insulare*, and *L. longiflorum*, var. *takesima*, and *L. Batemannia*, but no other host plants are known.

Geographical Distribution. Lilies affected with this virus were observed in Washington, U.S.A., in 1915, and it is thought that affected bulbs were exported from there to Bermuda, where the disease became of serious economic importance. It has been recorded from Java. There also appear to be a few records of the

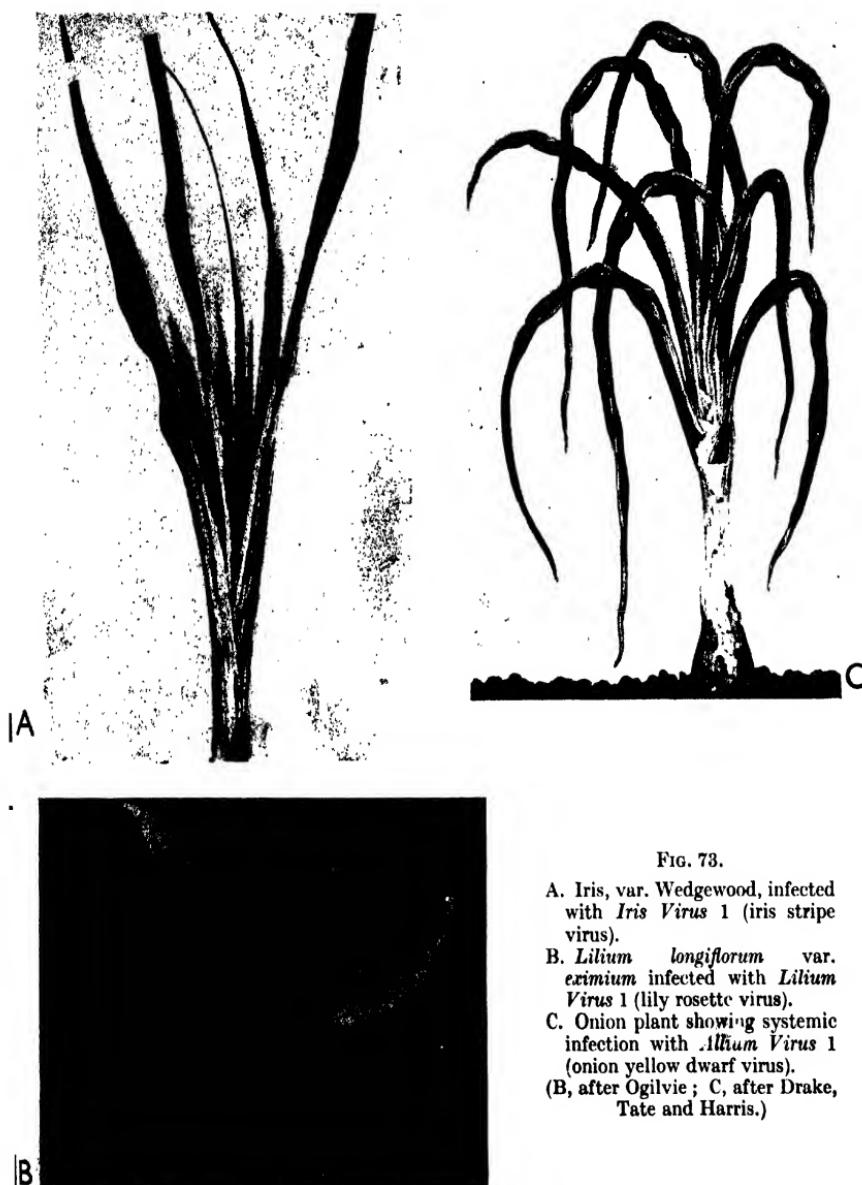


FIG. 73.

A. Iris, var. Wedgewood, infected with *Iris Virus 1* (iris stripe virus).
B. *Lilium longiflorum* var. *erimium* infected with *Lilium Virus 1* (lily rosette virus).
C. Onion plant showing systemic infection with *Allium Virus 1* (onion yellow dwarf virus).
(B, after Ogilvie; C, after Drake, Tate and Harris.)

disease in England and other parts of Europe, where the virus has been brought in on imported bulbs.

Control. The best method for the control of the disease under field conditions seems to be regular inspection and careful roguing of the crop. In addition, the aphis vector should be kept down by dusting or spraying with a nicotine insecticide. No discarded bulbs should be left lying about, nor should patches of lilies be left undug.

ALLIUM VIRUS 1. Melhus

Synonyms. *Onion Yellow Dwarf Virus*, Drake, Harris and Tate, 1932 ; *Onion Crinkles Virus* ; *Onion Mosaic Virus*.

The Virus

Thermal Death-point. The virus is inactivated after ten-minute exposures to a temperature between 75° and 80° C. Low temperatures do not affect the virus.

Dilution End-point. The virus loses infectivity at dilutions above 1 : 10,000.

Resistance to Ageing. The virus is inactivated after ageing for 100 hours in infective sap at a temperature of 29° C. Sap extracted from infected onion leaves which have been stored in the open for 100 hours at 29° C. is uninfective.

Transmission. *Allium Virus 1* is sap-transmissible, but there is no evidence that infection is carried in the seed or soil. There appear to be a large number of aphis vectors, of which the most important are *Aphis rumicis* L., *Aphis maidis* Fitch. and *Rhopalosiphum prunifoliae* Fitch. (Chapter VIII). According to Drake *et al.* (11), more than fifty aphis species have been shown experimentally to transmit *Allium Virus 1*. Among these may be mentioned the following :

Amphorophora rubi Kalt.

Aphis gossypii Glover.

A. pomi De G.

A. rumicis L.

Brevicoryne brassicae L.

Hysteroneura setariae Thomas.

Macrosiphum gei Koch.

M. pisi Kalt.

Myzus persicae Sulz.

Rhopalosiphum pseudobrassicae Davis.

Most of these aphides are described in Chapter VIII. The

aphides are capable of conveying infection to healthy onions almost immediately after having fed on a diseased plant. They also lose rapidly the power to infect if not again colonised on a diseased onion plant. These facts, added to the apparent capacity of any aphis species to act as vectors, suggest that the aphides are only mechanical vectors of the virus (10).

Differential Host

Narcissus jonquilla L., the jonquil, is susceptible to infection with this virus, the symptoms produced being similar to those on the onion plant.

Diseases caused by Allium Virus 1

Liliaceæ

Allium Cepa. The onion. *Yellow dwarf disease* (see Fig. 73, C). The first sign of infection in plants grown from naturally infected onion bulbs is a series of short yellow streaks which appear at the base of the first leaf emerging through the neck of the bulb. Onion plants inoculated in the leaves, either by needle or aphis, show these first symptoms only at the base of the leaves emerging after the inoculation. In general, all leaves emerging after the appearance of the first symptoms show signs of the disease, and those previously developed remain apparently healthy. Under conditions favourable for the development of yellow dwarf disease the leaves showing the short yellow streaks at the base become yellow throughout and also crinkled and somewhat flat. In this condition the leaves fall over and present an abnormal appearance. Flower stalks of infected plants producing seed show yellow streaks extending upward from the base. Later the streaks coalesce, the stalk becomes yellow throughout and twists and curls in a characteristic manner. The yellow, crinkled and drooping leaves and the twisted curled flower stems of infected plants give a decidedly dwarfed appearance. Bulbs of onion plants produced from infected sets are under-developed and of little commercial value, although they are usually well shaped. Bulbs from onion plants with only the last few leaves showing symptoms, or from plants with completely masked symptoms, are usually normal in development and appearance. The flower clusters of infected mother onion plants are smaller and have fewer flowers than normal plants. Masking of the symptoms of yellow dwarf disease occurs quite commonly in infected onion

plants, and such plants, while making normal growth themselves, are a source of infection to other healthy onion plants. Bulbs, from onion plants which mask the symptoms, when regrown produce infected plants that show symptoms (20).

Host Range. In addition to the onion, the following plants are susceptible to *Allium Virus 1*, *Allium sativum* L., shallot; *Narcissus tazetta* L., Chinese sacred lily; and *Narcissus jonquilla* L., the jonquil.

Geographical Distribution. The yellow dwarf disease was first observed in 1927 in Iowa, U.S.A. It has since been recorded from West Virginia, California and Minnesota. In 1929 Biemer reported an apparently similar disease of onions in Germany.

Control. The control methods practised in the United States of America against this disease are (a) indexing, (b) the production of virus-free stocks of bulbs in areas where the disease is absent, and (c) roguing out infected "volunteer" onion plants.

The method of indexing consists in regrowing sample lots of sets and mother bulbs in greenhouse beds or water culture under conditions whereby the percentage of infected bulbs in each lot can be determined.

As regards resistant varieties of onions, only one out of thirty-six varieties tested, Riverside Sweet Spanish, seemed to show any degree of resistance to the virus.

Spraying to kill the *aphis* vectors has not proved to be an efficient method of control.

IRIS VIRUS 1. Brierley and McWhorter

Synonyms. *Iris Mosaic Virus*; *Iris stripe Virus*.

The Virus and its Transmission. There is at present no information on the properties of this virus. It is transmissible by mechanical methods of sap-transfusion, but only with difficulty. The most successful methods of artificial transfer of the virus are by the insertion of a wedge of diseased stem tissue and by injection of diseased sap by means of a fine hypodermic needle. In the former method a wedge-shaped sliver is cut from the stem of the affected plant and inserted into a simple longitudinal slit in the stem of the healthy plant. The wound is then covered with a wrapping of raffia or thin rubber tape. To infect a healthy plant by sap-injection the internode is the most effective site for inoculation.

The insect vectors of the virus are the aphides *Macrosiphum gei* and *Myzus persicae* (see pp. 582 & 538).

Differential Host

The virus does not appear to infect plants of any other genus but the Iridaceæ. The English variety of bulbous iris "Wedgewood" is, however, very susceptible to infection and is useful as a test plant for the virus.

Diseases caused by Iris Virus 1

Iridaceæ

Iris filifolia Boiss., *I. tingitana* Boiss., *I. xiphium* L. Bulbous irises. *Stripe*. The disease in irises caused by this virus is known as stripe or mosaic and the symptom picture varies greatly according to the species and variety of iris affected. Symptoms in the bulbous iris include general dwarfing of the plant, mottling of the leaves, and "breaking" of the flower colours. The commercial importance of the disease lies chiefly in the dwarfing effect, which results in a shorter flower stalk for cut blooms. When affected plants are in bud or flower, the mosaic mottling of light green and bluish shades is readily seen and is especially prominent on the leaf bases and bud sheaths. The bud sheath is characteristically marked with bluish-green blotches on a pale green ground, or, less commonly, with yellowish streaks. On immature plants, mosaic appears as a yellow streaking of the leaves. Leaf symptoms are similar in varieties of bulbous irises of all classes, including all flower colours, but the flower symptoms vary. In plants affected with mosaic the younger leaves and bud sheaths are always more plainly mottled than the older (see Fig. 78, A).

Flower "breaks" are usually darker than the normal colour of the flower. For example, "broken" flowers of blue Imperator develop nearly black marks on a blue ground; those of white "D. Haring" show a purple mottling on the normal white, and those of lavender "Therese Schwartz" exhibit prominent purple blotches. The most characteristic flower marking consists of a series of dark blotches of "tear drop" design, the tail of the drop extending towards the throat of the flower. Feather-like cleared streaks, lighter than the normal flower colour, are associated with mosaic in yellow varieties (4).

Symptoms in Non-bulbous Irises (*Iris tuberosa*). The symptoms are essentially the same as those described for bulbous irises. The thinness of the blade-like leaves renders the pale areas more evident, especially by transmitted light.

Histopathology. Two main tissue changes usually accompany external symptoms : (1) reduction in number of chlorenchymatous cells and mature chloroplasts in areas beneath the epidermis where extreme mottling is visible, and (2) reduction in size of the epidermal cells of leaves that have been materially shortened by mosaic infection. The reduction in number of chlorenchyma cells and chloroplasts is not always apparent, but the reduction in size of epidermal cells is very constant and can be easily demonstrated by comparing the epidermal peelings from healthy and from diseased plants. Intracellular inclusions, or X-bodies, do occur, though they are not abundant ; two types of inclusion, a reticulate and vacuolate type, have been observed.

The epidermis of *Iris tingitana* is especially suitable for the study of the effect of mosaic on the chondriome. On fixation with Nemec's compound (formol, potassium bichromate, and chromic acid) and staining with acid fuchsin, the epidermal cells of mosaic leaves can be shown to contain mitochondria in a state of active division into granules, while the vacuome simultaneously breaks up into a number of vacuoles. At a more advanced stage of infection the mitochondria occur in groups surrounding an aggregation of small vacuoles. The cytoplasm in which the groups of mitochondria occur stains more and more deeply, so that eventually the latter cannot be distinguished. These areas form the intracellular inclusions, or "X-bodies" (15).

Host Range of Iris Virus 1. The virus appears to be confined to the Iridaceæ, and all commercial varieties of bulbous iris which have been tested have proved to be susceptible. Cross inoculations between different coloured varieties of bulbous irises and between varieties of Dutch and Spanish irises are readily successful. The virus is not transmissible to such Solanaceous test plants as the tobacco, tomato or petunia. It cannot be transmitted to tulips, nor can the tulip virus (*Tulipa Virus 1*) be transmitted to iris.

Geographical Distribution. The distribution of *Iris Virus 1* seems to be wide and is probably found wherever the iris is grown. The disease is common in Europe; it has been recorded from Holland, Bulgaria, France and England, and in the last-named country it seems to be on the increase. It is also widespread in the United States of America.

Control. The only methods of control at present available are the careful roguing of mottled plants and routine spraying or dusting to destroy the aphis vectors (4).



FIG. 74.
A. Freesia infected with *Freesia Virus 1*: note the white necrotic patches on the leaves.
B and C. Narcissus bulb and leaves showing symptoms of narcissus stripe, possibly caused by *Tulipa Virus 1*.

FREESIA VIRUS 1. Woodward

Synonym. *Freesia Mosaic Virus.*

The Virus and its Transmission. Very little is known of this virus, but it appears to be perpetuated by the corms. In some unpublished work, Woodward suggests that the insect vector may be the aphid *Myzus convolvuli* Kalt.

Disease caused by Freesia Virus 1**Iridaceæ**

Freesia sp. *Mosaic* (see Fig. 74, A). Symptoms appear on the young leaves not long after they are produced and increase in severity as the plants mature. On the leaves small water-soaked spots first develop, these areas subsequently dry up, leaving a white paper-like spot on the infected leaves. In a severe infection these paper-like areas coalesce and completely kill and dry up the leaves. The symptoms are visible on the majority of the leaves of a diseased plant, but a proportion of the leaves may remain free of any signs of disease.

Rust-coloured sunken areas have been observed in corms from affected plants, but the precise connection of these areas with the virus disease has not yet been shown.

The flowers are distorted, there is often extension of the chlorophyll into the petals and in some cases the blooms fail to open (Woodward).

Geographical Distribution. The disease was first observed in England by Woodward at Abingdon, in 1929; since then it has been recorded by many observers in different parts of England, where it now seems to be widely distributed. What is probably the same disease has also been recorded from Italy.

Control. Control measures are restricted to the eradication of infected plants and fumigation to destroy the insect vector.

SACCHARUM VIRUS 1. Brandes

Synonyms. *Sugar Cane Yellow Stripe Disease (Virus)*, Wakker and Went, 1898; *Sugar Cane Mottling Disease Virus*, Stevenson, 1917; *Sugar Cane Mosaic Virus*, Brandes, 1919; *Grass Mosaic Virus*, Brandes and Klaphaak, 1928; *Sugar Cane Virus 1*, J. Johnson's classification.

The Virus

Resistance to Chemicals. According to Rafay (44) the virus is easily inactivated by various chemicals. The following reagents destroy its infective power. copper sulphate, 1 : 500; hydrochloric acid, 1 : 1,000; mercuric chloride, 1 : 1,000; nitric acid, 1 : 800; sodium chloride, 1 : 25; hydrogen peroxide, 1 : 25; and formalin, 1 : 50. Zinc powder and manganese dioxide, 1 : 20, did not affect the virus.

Serological Reactions. In serological tests with anti-sugar cane mosaic and anti-healthy juice sera in which rabbits were immunised by intravenous inoculation with Chamberland candle filtrates of mosaic and healthy leaf juice, the anti-mosaic serum inactivated the mosaic leaf juice, while the anti-healthy serum had no effect. Precipitation tests with the anti-mosaic serum gave a positive reaction with mosaic leaf juice and a negative one with healthy leaf juice, the anti-healthy serum being faintly positive to both (9).

Thermal Death-point. The virus appears to be inactivated by a temperature of 53° to 54° C.

Dilution End-point. The virus tolerates dilution of 1 : 10 only; it loses infectivity at a dilution of 1 : 100 (44).

Resistance to Ageing. Expressed infective juice of the sugar cane tends increasingly to lose its infectivity when kept exposed to the air for a day or more at room temperatures or even as low as 4° C. Juice retains infectivity for twenty-seven days if kept frozen at about — 6° C. (32). Rafay (44) finds that the virus loses potency in expressed sap in two hours.

Filterability. Desai (9) used a filtrate from a Chamberland candle for his serological experiments, but Rafay (44) states that the virus will not pass an L₃ Chamberland candle, and, furthermore, that it is held back by ordinary filter paper.

Transmission. *Saccharum Virus 1* is transmissible by mechanical methods, but only with difficulty, and various inoculation techniques have been designed to increase the percentage of successful transmissions. In one method a drop of inoculum from young mosaic suckers is deposited with a pipette in the wedge-shaped opening on healthy canes between the youngest expanded leaf blade and the next younger leaf on the same side of the leaf spindle; a very fine needle set in a glass rod is then passed horizontally, or somewhat obliquely, downward through the liquid and into the submerged area of the still rolled leaf, several cuts with the needle being made through the leaf tissue in order to



FIG. 75. *Saccharum Virus 1* (sugar cane mosaic virus).

- A. Leaf of healthy sugar cane.
- B. Leaf of mosaic sugar cane.
- C. Leaf of mosaic maize (*Saccharum Virus 1*).
(A and B, after McClean ; C, after Storey.)

allow of contact of the virus with the severed fine transverse connections of the vascular bundles (32). Another method of mechanical inoculation consists of stripping the central spindle of the outermost leaf, binding the cylinder of tender leaves thus exposed with a mosaic leaf and pricking through this with a number of insect pins mounted in a handle (45).

There is no evidence of seed or soil transmission of this virus. The chief insect vector is the corn aphid, *Aphis maidis* Fitch., while the rusty plum aphid, *Hysteroneura setariae*, is apparently also an occasional vector (see pp. 520 & 530) (22).

Differential Host

Holcus sorghum L. Sorghum grass is extremely susceptible to *Saccharum Virus 1* and makes a useful test-plant.

Diseases caused by Saccharum Virus 1

Graminaceæ

Saccharum officinarum L. Sugar cane. *Sugar cane mosaic* (see Fig. 75, A and B). The primary and critical symptom of this disease is the appearance of pale patches or blotches in the green tissues of the leaves. The blotches themselves are primarily of a uniform green throughout, but at the same time they are several shades lighter than the normal green tissues which surround them. They are not constant in size or shape even on the same leaf, although they may be large in some varieties and small in others. They are usually irregularly oval or oblong in outline, their longer axes lying parallel to the mid-rib. They are not confined between veins, and consequently are not of uniform width throughout any considerable part of their length. They do not simulate stripes.

In all cane varieties the characteristic light patches on diseased leaves are very distinct when the leaves first unroll from the spindle. The newly opened leaves are, therefore, the ones to examine when seeking symptoms of the disease. The appearance of light blotches on the leaves is the primary and critical symptom of mosaic and the one upon which a diagnosis should be based.

Some varieties of sugar cane may carry the virus year after year without showing any symptoms other than the mottling of the leaves, while other varieties may display decided secondary symptoms. Mottling or marbling of the stem is the one secondary symptom most frequently displayed by diseased canes. This mottling is not shown by all varieties, but it is very pronounced in some, especially after exposure of the stem to strong sunlight. In

some cases the mottling of the stem or rind produces death of the tissue, which in turn gives rise to cankered areas. The production of small and deformed sticks is a symptom of the disease in a few varieties, and in sticks long affected with the virus there may sometimes be found irregular masses of internal tissue, more or less stained or discoloured. The disease does not as a rule induce an appreciable change in the size or shape of the leaves, but in certain varieties the stunted shoots sometimes terminate their efforts to grow by throwing a few twisted or distorted leaves in the manner characteristic of canes about to succumb to Fiji disease (*Saccharum Virus 2*, p. 432) (29).

Histopathology. The cells of affected leaves always show, in one part of the cytoplasm, an area of proteolysis. This area is recognisable under the microscope as one more heavily stained than the rest and consisting of a vacuolated mass characteristic of the usual appearance of X-bodies (12).

Host Range of *Saccharum Virus 1*

A number of different species of wild and cultivated grasses, etc., are susceptible to infection with the sugar cane mosaic virus. The following are some of the more important of these susceptible plants :

Cultivated Crops and Grasses

Sugar cane	<i>Saccharum officinarum.</i>
Maize or corn	<i>Zea mays</i> (see Fig. 75, C).
Sorghum	<i>Holcus sorghum.</i>
Pearl millet	<i>Pennisetum glaucum.</i>
Sudan grass	<i>Andropogon sorghum sudanensis.</i>
Wonder forage grass	<i>Andropogon</i> sp.
Tunis grass	<i>A. sorghum virgatus.</i>
Eulalia	<i>Misanthus sinensis.</i>
Guatemala grass	<i>Tripsacum laxum.</i>

Wild Grasses

Wild sugar cane	<i>Saccharum narenga.</i>
Bull-grass	<i>Paspalum boscianum.</i>
Crab-grass	<i>Syntherisma sanguinalis.</i>
Yellow foxtail	<i>Chætochloa lutescens.</i>
Giant foxtail	<i>C. magna.</i>
Barnyard grass	<i>Echinochloa crusgalli.</i>
Panicum	<i>Panicum dichotomiflorum.</i>

Brachiaria . . .	<i>Brachiaria platyphylla.</i>
Bristly foxtail grass . . .	<i>Chætochloa verticillata.</i>
Goose grass . . .	<i>Eleusine indica.</i>
S. African wild grass . . .	<i>Setaria sulcata</i> (8, 61).

On all these grasses the symptoms of infection are a mosaic mottling of the same type as that which develops on infected sugar cane.

Geographical Distribution. It is generally agreed that the virus of sugar cane mosaic originated in the East, and has been distributed from there in sugar cane cuttings to practically all the sugar cane growing countries of the world. It has been recorded from the following places : Barbados, Cuba, India, Jamaica, Philippines, Porto Rico, Queensland, South Africa, Surinam, Uganda, United States of America, Argentine, New Guinea, Hawaii, Trinidad, Egypt, Kenya, Madagascar, British Guiana, Peru, Brazil, Dutch East Indies, and Venezuela.

Control. The main methods of control for sugar cane mosaic may be outlined as follows : (1) The use of resistant varieties of sugar cane ; (2) systematic roguing of infected canes ; (3) the use of selected healthy setts ; (4) the elimination, where this is practicable, of wild grasses in the neighbourhood of the canes ; and (5) legislation. The work of breeding mosaic-resistant sugar canes is probably the most extensive and successful ever carried out. The species of *Saccharum* which were used in this work are : *S. officinarum*, thick tropical, this is susceptible to mosaic : *S. spontaneum*, wild, resistant to mosaic ; *S. barberi*, thin, North Indian, susceptible to mosaic ; *S. sinense*, medium thick, India and China, both resistant and susceptible to mosaic ; *S. robustum*, New Guinea, resistant to mosaic. The early Javan work was in breeding for resistance to Sereh disease (*Saccharum Virus* 3, see p. 486) by crossing Chunnee (*S. barberi*) with *S. officinarum*. This produced the varieties P.O.J. 86, 218, 228, 284. These were on the whole exceedingly susceptible to mosaic infection and produced plain symptoms, but their growth was hardly affected, i.e., they were highly "tolerant" of the virus. They were the chief agents of the almost world-wide distribution of mosaic before it was recognised. In later work Javan breeders concentrated upon the cross of Kassoer (a resistant variety believed to be a natural cross between *S. spontaneum* and *S. officinarum*) and *S. officinarum*. This produced the early 2,000 numbers, particularly P.O.J. 2,864. Crossing this with *S. officinarum*, they

produced the commercially important and very highly resistant varieties P.O.J. 2,714, 2,725, 2,727, etc. Recently P.O.J. 2,725 has been widely used for further crossing with *S. officinarum* in Java, Porto Rico, Barbados, etc., and has produced a wide range of highly resistant varieties. The surprising feature of this work is the continued appearance of the factor for resistance inherited from so far distant an ancestor (Storey *in litt.*). Resistant varieties of sugar cane are now in use in most of the sugar cane growing countries. Thus in Uganda and Porto Rico good control of the disease has been achieved by the use of P.O.J. 2,725 and 2,878; in India the varieties are P.O.J. 2,878, 2871 and 2,696; in Réunion, these three and P.O.J. 2,725, 2,714 and C.O. canes 214, 281, 290; in Tucumán, Argentine, P.O.J. 36 and 213, and in South Africa the same varieties and C.H. 64/21. In the last-named country the situation is complicated by the wide distribution of the virus in the wild grass *Setaria sulcata*.

In Réunion legislation has been introduced to make compulsory the eradication of the most susceptible canes and to prohibit the planting of varieties of sugar cane, for the reconstitution of plantations, other than those supplied or recommended by the competent agricultural authorities.

In badly affected areas the only possibility of control consists in the complete replacement of the diseased canes by resistant varieties.

Strains of *Saccharum Virus 1*

There is little doubt that this virus, like several others, exists in a number of closely allied strains. Since no investigation has been carried out so far upon the question of strains of *Saccharum Virus 1*, the writer has made the following tentative arrangement, based solely upon descriptions in the literature of the subject, of what appear to be strains of this virus.

SACCHARUM VIRUS 1A. Storey

Synonym. *Transvaal Grass Mosaic Virus*.

This virus was observed affecting maize and *Sorghum* spp. in the Transvaal, South Africa. The symptoms it produced on these plants were not distinguishable from those produced by the ordinary sugar cane mosaic virus. Attempts to transmit this virus to sugar cane, however, by means of the insect vector *Aphis maidis*

and by mechanical inoculation methods were all unsuccessful. It is concluded, therefore, that this virus is non-virulent to sugar cane (51).

SACCHARUM VIRUS 1B. Summers

On sugar cane variety, Canal Point 28/60, this strain is characterised by a slight mottling with very little chlorosis and no noticeable stunting. On a selected range of sugar cane varieties it produces the ordinary mosaic symptoms typical for the particular variety. The varieties are Louisiana Purple, C.O. 281, P.O.J. 36-M., P.O.J. 213, and P.O.J. 234. This strain has only been obtained from four C.P. sugar cane seedlings in Louisiana, U.S.A.

SACCHARUM VIRUS 1C. Summers

On C.P. 28/60 this strain produces a severe mottling with large chlorotic areas, a varying extent of necrosis, and marked stunting. It gives rise to ordinary mosaic symptoms on the other varieties, as in the case of *Strain 1B*.

SACCHARUM VIRUS 1D. Summers

Virus 1D becomes evident on C.P. 28/60 by the development of elongated, almost white, blotches or islands, some of which later coalesce into long, yellowish-white streaks, often running the full length of the older leaves. The streaks may follow the mid-rib, and are frequently accompanied by necrosis, sometimes so severe as to produce temporary blighting, or occasional death, of the growing point. Severe symptoms similar to the above appear also on Louisiana Purple and the four other varieties mentioned.

Creole maize is susceptible to this strain, but develops different symptoms from those on the sugar cane; instead of the bold white stripes and early necrosis seen on the latter host, the maize plants exhibit a fine mottling of the inner leaves. In about a week, however, these slight symptoms fade and the plants wilt and die.

SACCHARUM VIRUS 1E. Summers

Strain 1E produces identical symptoms on C.P. 28/60 to those produced by *Strain 1D*, but it gives rise to only ordinary mosaic symptoms on the five other varieties (58).

SACCHARUM VIRUS 1F. Tims

This strain is a yellow-type virus causing very severe injury to the sugar cane C.P. 28/70 in Louisiana. A very conspicuous yellow mottling is produced, together with a decrease in the size of the leaves. This yellow strain is transmissible to other varieties of sugar cane, on which it gives rise to mild symptoms characteristic of the type virus (59).

SACCHARUM VIRUS 1G. Storey

Synonym. "Agaul" *Mosaic Virus*.

A sugar cane variety, Agaul, imported to Natal from India many years ago, is now found, both in South and East Africa (whither it was imported from Natal), entirely affected with a mosaic disease. The pattern shown by the young leaves of affected canes is quite typical of the mild symptoms due to infection with the type virus. Many attempts to transfer this virus by needle-inoculation and by means of *Aphis maidis* have all failed. Nor is there any evidence that natural spread to other varieties occurs in the field. On this evidence Storey (55) tentatively separates this virus from the common mosaic group, but the writer has included it here for convenience, and until further investigation enables it to be placed in its rightful category.

SACCHARUM VIRUS 2. Kunkel

Synonyms. *Sugar Cane Fiji Disease Virus*, Muir, 1910, Kunkel, 1928, Ocfemia, 1982, Mungomery and Bell, 1988.

The Virus and its Transmission. Nothing is known at present of the physical and other properties of the virus. It is apparently not sap-inoculable, although many different methods of mechanical transmission have been tried, including those which are successful in transmitting *Saccharum Virus 1*. There is no evidence of transmission through the seed or soil. Two insect vectors have been described, one in Queensland, *Perkinsiella saccharicida* Kirk (see p. 494) (89), and the other in the Philippine Islands, *Perkinsiella vastatrix* Breddin (41). It is possible that other species, *P. vitiensis*, for example, may also be able to act as vectors. There is a certain amount of evidence, as regards *P. saccharicida*,



FIG. 76. *Saccharum Virus 2* (causing Fiji disease).

A. Infected sugar cane showing stunted malformed top typical of later stages of the disease, var. E.K.I.

B. Infected leaf of sugar cane showing leaf galls, var. Malabar.

C. Cross-section of diseased leaf sheath ; in the gall shown the sclerotic tissue completely surrounds the infected cells.

(A and B, after Mungomery and Bell ; C, after Kunkel.)

that the adult insects are unable to pick up the virus *de novo*, but must have fed as nymphs, in the first place, upon diseased sugar cane.

Differential Hosts

Saccharum Virus 2 seems to be confined in its host range to the sugar cane.

Disease caused by *Saccharum Virus 2*

Graminaceæ

Saccharum officinarum. Sugar cane. *Fiji disease* (see Fig. 76). The one critical symptom by which Fiji disease may be recognised is the occurrence of elongated swellings or galls on the under surface of the leaves (see Fig. 76, B). These galls extend along the larger veins or vascular bundles and are, in fact, formed by the abnormal growth of the tissues comprising these bundles. Galls are produced in similar manner in the vascular bundles of the stem and may be detected by splitting open the stick of an affected shoot. Galls of this nature are not induced by any other known cane diseases, and consequently their presence on the leaves or in the stem of a cane plant may be accepted as conclusive evidence that the plant in question is affected with Fiji disease.

The most conspicuous symptom of Fiji disease to be noted in the field is a shortening and crumpling of the last leaves to unfold from the spindle. This peculiarity will attract attention when the observer is still a considerable distance from the affected cane. A diseased shoot may attain a fair length and be clothed with many healthy looking leaves of the usual length and colour, but suddenly it loses power to produce normal leaves, throws out a few bent and twisted stumps and then ceases to grow altogether. The stick may remain alive for months or it may soon die. When such a stick is examined the characteristic galls are usually to be found on most of the healthy looking leaves which are not otherwise distorted and on all of the deformed aborted leaves. These latter leaves look as if they had been burned or scalded before expanding, the injury destroying the upper half or two-thirds of the leaf blades, leaving short crumpled stumps. The disease is cumulative in the cane, the galls mark a well-advanced stage of the disease, and the distortion of the apical leaves is its final culmination (29).

Histopathology. Examination of the tissues of terminal buds of diseased stalks shows that groups of infected cells frequently

occur a short distance back of the growing point. Here, in the phloem or in the tissues immediately adjoining the phloem, may be found small groups of cells that contain deep-staining, spherical or oval-shaped, intracellular inclusions, or X-bodies.

Successive stages in the formation of the galls can be found in the young stalk tissue at different distances back from the growing point and in the young leaves of different ages. The earliest stages observed consist of very small groups of infected cells just below the growing point in the tissues that are in process of differentiating into vascular bundles; the galls then gradually spread longitudinally along the bundles. The galls always originate in the phloem and are caused by the proliferation of the phloem cells and occasionally of cells in the surrounding tissue. This explains why the galls are found on the under but not the upper surface of the leaf. As compared to healthy, or non-gall, tissue, the cytoplasm of the gall cells is more dense and stains deeply and the line of demarcation between the normal and abnormal tissue is very sharply defined (23, 39) (see Fig. 76, C).

Geographical Distribution. Fiji disease of sugar cane takes its name from the British Crown Colony in which it was first observed about the year 1906. The disease has since been recorded from New South Wales, Java, the Philippine Islands and New Guinea.

Control. In Queensland control of the disease can be effected by plant selection and the careful roguing of diseased stools as soon as they are detected in the young cane. The two most susceptible varieties are M. 1,900 seedling and D. 1,135, and in some localities where the stocks are generally infected their elimination is recommended. No variety is known to be immune to *Saccharum Virus 2* and none can be classed as highly resistant. North has classified the chief varieties of sugar cane into five groups, ranging from moderately resistant to susceptible, as follows :

- (1) Badila (N.G. 15), Bogela (Nanemo), Mahona (N.G. 22), Pompey (7 R. 428), Q. 818, Rose Bamboo.
- (2) Innes 181, N.G. 16.
- (3) H. 109, M. 1,900 Seedling, Yellow Caledonia (Malabar).
- (4) D. 1,135, H.Q. 285 (in Fiji), N.G. 23, N.G. 24.
- (5) B. 208, Daniel Dupont, N.G. 14.

A complication has arisen in the control of Fiji disease in Queensland owing to the fact that the commercially important varieties of the Kassoer type, which are much grown because of

their resistance to gumming disease, are found to be extremely susceptible to this virus (89).

In Fiji itself good control has been achieved by the use of the more resistant varieties, by the use of cuttings from healthy canes only for purposes of propagation and by the replanting of all fields after taking only one ratoon crop.

SACCHARUM VIRUS 3. Kunkel

Synonym. *Sugar Cane Sereh Disease Virus*, Wakker and Went, 1898, and Kunkel, 1928.

The Virus and its Transmission. Nothing is known of the properties of this virus nor of its mode of transmission in the field. It is not sap-inoculable, but is carried in the cuttings.

Disease caused by Saccharum Virus 3

Graminaceæ

Saccharum officinarum. Sugar cane. *Sereh disease*. "Sereh" is the Javanese name for lemon grass (*Andropogon Schœnanthus* L.), and this name was applied to the cane disease because in affected plants the growth of the cane stool is arrested and the stool thereby converted into a bushy tuft of leaves somewhat resembling the tufts of lemon grass. This symptom, however, is by no means a constant feature of the disease in all varieties. When the disease first appeared in Java the Cheribon cane was the one standard variety grown throughout the island, and when affected this variety showed the following symptoms to a greater or less extent :

(1) Inability to grow ; the majority of shoots remaining short and stunted.

(2) The vascular bundles in the sticks are coloured red, due to the presence of a red, gummy substance in the vessels.

(3) Pronounced growth of adventitious roots under the leaf sheaths from many or all of the nodes on diseased sticks.

The last-named symptom is a frequent manifestation in some cane varieties, but perhaps the most reliable symptom of infection is the presence of red gum at points in the vascular tissue of the stem. Stunting of the stool is also caused in some varieties (29).

Geographical Distribution. Since the first appearance of sereh disease in Java, it has been reported from Borneo, Sumatra, Malakka, India, Mauritius, Australia, Fiji, Formosa, Hawaii, and Ceylon.

Control. The chief methods of control for sereh disease are the use of resistant varieties, the use of cuttings from healthy canes only for purposes of propagation and the abandonment of the practice of ratooning.

In Java the disease has come to be of little importance owing to the use of the resistant variety of sugar cane, P.O.J. 2,878.

SACCHARUM VIRUS 4. Storey

Synonym. *Sugar Cane "R.P. 8" Streak Virus*, Shepherd, 1929.

The Virus and the Disease it causes. A number of years ago a streak disease of sugar cane was reported in Mauritius. It occurred only in one variety, R.P. 8, and spread to other plants of this variety, but to no other kind. The symptoms in R.P. 8 cane are very similar to those of streak disease in that plant caused by *Zea Virus 2*, but repeated attempts to transmit *Saccharum Virus 4* to maize, to healthy R.P. 8 cane and to other cane varieties, by means of *Cicadulina* spp. and *Peregrinis maidis*, have failed. For the present, therefore, this virus is placed in a separate category (55).

SACCHARUM VIRUS 5. Bell

Synonym. *Sugar Cane Dwarf Disease Virus*.

The Virus and the Disease it causes. Nothing is known of this virus or its natural mode of transmission ; it does not appear to be sap-inoculable. The symptoms of the dwarf disease in the sugar cane variety P.O.J. 2,714 are as follows : the leaves, particularly the younger leaves, of diseased canes are marked with fine longitudinal yellowish stripes. The stripes are usually short, $\frac{1}{2}$ to 2 inches long, but they may often be as much as 6 inches in length. They follow the direction of the veins and are about $\frac{1}{16}$ inch wide, but may run together to give moderately wide bands, especially at the margin of the leaves, and are not evenly distributed over the leaf surface as a rule. The leaves of diseased canes are stiff and erect, thus imparting a fan-like appearance to the cane top ; the spindle and the younger leaves are usually twisted and deformed, of a lighter colour and shorter than the normal. There is a progressive masking of the streaks in passing to the older leaves, which are of a darker green than normal. The most

striking symptom is seen in the case of primary infection. Here the stool consists of a number of stunted shoots which form no cane ; the leaves are erect, stunted and clustered, and bear the typical yellowish streaks with, as a rule, scalded or reddish tips and margins or stripes, and later becoming frayed and torn. This stage of the disease much resembles extreme cases of Fiji disease (*Saccharum Virus 2*) and sereh disease (*Saccharum Virus 3*). In cases of secondary infection, growth ceases suddenly and the top of the stalk tapers off to a point, forming the fan-like top. Such stunted stalks are soon outgrown by the healthy stalks in the same stool, and the upper internodes become sunken.

Histopathology. No definite abnormalities have been observed in either stems or leaves of plants in which the infection was secondary, but a marked derangement of the tissues occurs in the vascular bundles of the leaves of the extremely stunted plants which result from primary infection. The major bundle may be considerably enlarged, very irregular in shape and frequently fused with an adjoining minor bundle. The chlorophyll-bearing sheath is incomplete as a rule, and may be represented by very few cells or be entirely absent in extreme cases. Within the bundle there is an abnormal development of comparatively thin-walled lignified cells which frequently radiate through the bundle in two or more strands, bringing about distortion and altering the relative positions of component tissues. Phloem may be almost entirely absent and confined to one of the resultant sectors, or may be found scattered in more than one sector or at the ends of the lignified strands. The walls of the cells of the lignified inner sheath surrounding the phloem appear thinner than is the case in normal cane, and the sheath is completely disrupted, but whether the strands of woody cells have their origin in this particular tissue has not been established (2).

Storey (55) differentiates dwarf disease of sugar cane (*Saccharum Virus 5*) from streak disease of sugar cane (*Zea Virus 2*) by the following characteristics :

- (1) The stiff fan-like top occurs only in *young* streak diseased Uba cane, and in later growth diseased plants are not noticeably different in habit from healthy plants.
- (2) The stripes of streak disease are white rather than yellow.
- (3) They are usually only a few millimetres long and rarely more than an inch in length.
- (4) The leaf markings are evenly distributed over the leaf in the case of streak.

- (5) There is no deformity of the inner leaves, and in streak older leaves do not assume a darker green than normal.
- (6) There is no masking of streak symptoms in older leaves.
- (7) Such abnormally severe stunting has not been observed with streak, nor do shoots die prematurely.
- (8) Secondary infection causes no sudden cessation of growth.
- (9) Shrinking of internodes has not been noticed.
- (10) Ratoons grow normally after an initial stunted stage.

Geographical Distribution. The disease of dwarf seems to be restricted to Queensland, where it was first observed in 1930.

ZEA VIRUS 1. Kunkel

Synonyms. *Corn Mosaic Virus*, Kunkel, 1922; *Corn Leaf-stripe Virus*, Stahl, 1927; *Corn Virus 1*, J. Johnson's classification; (?) *Maize Stripe Disease Virus*, Storey, 1936.

The Virus and its Transmission. It does not appear to be possible to transmit this virus by mechanical inoculation and there is no information on the physical or other properties of the virus. The insect vector is the leafhopper *Peregrinus maidis* Ashm. (see p. 489).

Disease caused by Zea Virus 1

Graminaceæ

Zea mays L. Corn, maize. *Stripe.* The disease caused by this virus in maize has been known as "corn stripe" and "corn mosaic." There is considerable variation in the symptom picture o.. the different varieties of maize. As a rule the first visible symptoms are manifested by small elongated white specks usually on one side only of the mid-rib near the base of a young leaf. The specks elongate and spread out along the leaf parallel to the mid-rib, forming rather fine interrupted stripes. These spots may join up and form almost continuous stripes. As the disease advances the leaves become covered with slender interrupted stripes. Under certain conditions these stripes may be coarse and conspicuous, radiating from the mid-rib. In some cases the fine interrupted stripes fuse, causing the chlorophyll to disappear and form yellow bands on the leaves (48). Priode, in Cuba, has classified the symptoms into three main types. *Type I.* Stripes due to a whitening of the leaf veins both large and small, very fine and close together, usually extending the full length of the leaf, but in some

cases fading into short stripes or dots at varying lengths. In some cases all the veins in a leaf may be uniformly affected, while in others only a few veins on one or both sides become chlorotic. In extreme cases affected tissues become necrotic. Stripes may be present on sheaths, ear-husks and stalks. *Type II.* The symptoms of this type appear as distinct, coarse, parallel stripes due to whitening of the large leaf veins, the smaller veins and intervening tissues usually remaining green. As a rule stripes extend the full length of the leaf and they may show plainly on sheath, ear-husks and stalk. *Type III.* The symptoms show as broad chlorotic bands rather than distinct stripes, and both the veins and interveinal tissue are affected. The bands may extend the full length of the leaf, but usually fade into series of dots or stipple stripes at different lengths on the leaves. In some cases the entire leaf bases are affected from side to side, while in others one or more broad bands appear on one or both sides of the mid-rib. The bands sometimes follow along the mid-rib. The above are descriptions of the disease in Cuba. What is thought to be the same disease has been described in Trinidad (5). In this case also there appears to be considerable variation in symptoms. In some varieties affected leaves wither from the tip backwards and from the margin inwards, the lighter coloured tissue being first affected and turning either a dead brown, or, in some instances, passing through a preliminary deep red or purplish-red phase of colour.

Histopathology. Sections through the red areas on the leaves show that the discolouration is associated with changes in the chloroplasts. The plastids in the palisade cells of the discoloured tissue may be seen to be broken down into granular masses. The walls of some of the mesophyll cells show signs of dissolution (5).

Cook (8) describes phloem necrosis as an internal symptom of the stripe disease of corn. This necrosis is accompanied by a thickening of the walls of the epidermal cells, the fibrous cells and sheath. In some cases these cells develop thick walls without phloem necrosis. The effect of the disease on the cell contents is very noticeable. The chloroplasts in the sheath cells of the bundles with necrosis and the parenchyma cells around such bundles are few in number, while the corresponding cells in a section from a healthy plant show large numbers of chloroplasts. Intracellular bodies have not been observed.

Host Range. *Zea Virus 1* is apparently not transmissible to sugar cane, but in Trinidad it occurs naturally on Sorghum, to

which it has also been transmitted experimentally by means of the insect vector (*P. maidis*).

Geographical Distribution. Corn stripe disease has been recorded from Cuba, Hawaii, Trinidad, probably Tanganyika, Mauritius and Porto Rico.

ZEA VIRUS 2. Storey

Synonyms. *Maize Streak Disease Virus*, Storey, 1925; *Corn Virus 2*, J. Johnson's classification.

The Virus

It is fairly clear from the work of Storey and McClean that there exist more than one streak virus affecting maize; these viruses are, however, very closely allied and may legitimately be considered, at all events for the present, as strains of *Zea Virus 2*. These various strains of the virus seem to be specialised, each one for a particular host (57).

Since the virus is not inoculable by mechanical means, there is little information on its properties.

Filterability. Experiments have been carried out in the feeding of the leafhopper vector through membranes upon the juice of streak diseased maize plants after passage through various filters. Tests of these leafhoppers have shown that at a pH of about 6 the virus passes through Chamberland L₁, Berkefeld V and N filters, and less freely through Chamberland L₃. The virus does not pass a Seitz E.K. filter disc (52).

Transmission. *Zea Virus 2* is not carried in the seed, nor can it be transmitted by any mechanical method of inoculation. The insect vectors are three species of leafhoppers, *Cicadulina (Balclutha) mbila* Naude, *C. zea* and *C. nicholsi* (see pp. 474-476). Of these the first named is the most important vector. The relationship existing between this virus and the insect vector has been studied in detail by Storey (58, 54), and his findings are of great interest and importance. Races have been bred of the species, *Cicadulina mbila* Naude, which are, on the one hand, able, and, on the other, unable, to transmit the virus of streak disease in the natural process of feeding on maize plants. These races are termed "active" and "inactive" respectively. Further, the crossing of the pure races has demonstrated that the ability to transmit is inherited as a simple dominant Mendelian factor, linked with sex. Storey has also demonstrated that a simple

puncture of the abdomen with a sterile needle, either following or followed by a feed on a diseased plant, sometimes causes inactive individuals of *C. mbila* to become infective. By comparison of the efficacy of different positions for the puncture, it was concluded that the treatment was successful only if the needle had penetrated some part of the intestine. Inactive races of *C. zeæ* proved to be susceptible to inoculation with *Zea Virus 2* by the methods successful with *C. mbila*.

By the inoculation of the appropriate fluids, it was shown that the virus was present in infective active individuals of *C. mbila*, (a) in the contents of the rectum, if the insect had recently fed on a diseased plant, but not otherwise; (b) in the general contents of thorax or abdomen; and (c) in the blood, whether the insect had fed recently upon diseased or healthy plants. The virus was not found in the naturally voided faeces. The appearance of the virus in the blood preceded in time the development of the power to cause infections. Inactive races of *C. mbila*—normally unable to transmit the virus—were made infective by needle-inoculation with the virus. The proportion of successes was, however, significantly less than with active races. After a feed on a diseased plant the inactive insect was found to have the virus in its rectum, but never in its blood.

It is concluded from these observations that in active individuals of *C. mbila* the virus, entering the intestine by the mouth, passes through the intestinal wall into the blood; and that, in the inactive insect, the cells of the intestinal wall resist the passage of the virus. It is recognised that there may be some secondary mechanism of resistance; nevertheless, in many inactive individuals, once the barrier of the intestinal wall has been passed the virus behaves as in an active insect.

On emerging from the egg all insects are free of *Zea Virus 2*. If of a suitable race, they may thereafter pick up the virus by feeding, for as short a time as one minute, upon a chlorotic area of a maize leaf, but *not* on the intervening green area. They will not become infective at once, but only after a delay of a day or two. Once infective they will infect every plant upon which they feed for a day or two, and often for an hour; and they may remain infective through several months before they die. They may infect a maize plant by feeding upon any above-ground part of it. The plant shows no effect at the point where the insect punctures, but only on the young leaves that emerge some days after the insect's feeding.



FIG. 77.

- A. Leaf of maize plant affected with *Zea Virus 2* (maize streak virus) X1.
- B. Leaf of sugar cane (var. Black Tanna) naturally infected with *Zea Virus 2* X1½.
- C. Leaf of maize plant affected with *Zea Virus 8* (mottling disease virus) X1.
(A and C, after Storey ; B, after Storey and McClean.)

Differential Host

Zea Virus 2 is transmissible to a wild African grass *Digitaria horizontalis* Willd., on which the symptoms consist of numerous rather broad streaks on the leaves.

Diseases caused by *Zea Virus 2*

Graminaceæ

Zea mays L. Maize, corn. *Streak disease* (see Fig. 77, A). Streak disease, when fully developed in the maize plant, is characterised by a pronounced chlorosis of the leaves confined to narrow broken stripes arranged along the veins. The stripes vary from a few millimetres to several centimetres in length, and are individually from 0·5 to 1 mm. in width, although frequently fusing laterally to form wider composite stripes (see Fig. 78, E). The chlorotic tissue generally appears an opaque yellow when viewed by transmitted light, affording a marked contrast to the deep green of the remainder of the leaf. On some leaves a small proportion of the stripes may appear translucent, and in this case the tissue may be slightly shrunken. The stripes are nearly evenly distributed over every leaf formed since the plant became diseased.

The first sign of the disease in an individual plant is the appearance of nearly colourless spots, generally almost circular, about 0·5 to 2 mm. in diameter, upon the lowest exposed portions of the youngest leaves. These earliest spots are generally widely scattered, frequently separated by 2 or 3 cm. As more of each leaf becomes exposed by growth the youngest portions exhibit a progressive increase in the frequency of the spots, which now show a more or less elongation in the line of the leaf veins. At this stage they may be evenly distributed over the basal portion of the leaf or confined to tracts following groups of leaf veins. Soon, however, the spotting becomes general over the whole of the base of the leaf, and continues to appear at uniform frequency over all the new leaves subsequently formed. The chlorotic areas are delimited before the leaf unfolds, and no alteration in their size or shape occurs after the leaf has attained its full growth (50).

Saccharum officinarum. Sugar cane (see Fig. 77, B). The leaf of an affected plant of Uba sugar cane exhibits over its whole surface a pattern produced by broken, narrow, pale stripes running in the direction of the veins of the leaf. Each stripe is in width $\frac{1}{2}$ to $\frac{1}{4}$ mm. and nearly uniform, while in length it may vary from $\frac{1}{2}$ mm. to 1 cm. or more. Upon separate individual plants the stripes may be

crowded to a greater or less extent, but their frequency does not vary upon the different parts of a single leaf blade (49). On Uba cane the disease generally resembles the streak disease of maize, except that the chlorotic areas are narrower and more sparsely distributed upon the leaf surface. The virus from Uba cane is readily transmissible to Uba cane; when transferred to maize it produces in this species only a mild form of the streak disease, easily distinguishable from normal maize streak. *Zea Virus 2* is apparently incapable of causing permanent infections of sugar cane. There is little doubt that each host or group of hosts has a strain of the virus specialised to it. The strain of virus that produces the severest symptoms in maize is incapable of attacking sugar cane; on the other hand, as mentioned above, the sugar cane strain produces only a mild form of the disease in maize (57). Recently another strain apparently also specialised to maize has been recognised (A. P. D. McClean *in litt.*).

Host Range. It is thought that wild grasses and alternate hosts play only a small part in maintaining *Zea Virus 2*, though the two grasses, *Digitaria horizontalis* Willd. and *Eleusine indica* Gaert., are commonly affected.

The following twenty-two grasses, both wild and cultivated, have been observed in the field bearing chlorotic leaf markings resembling those of maize streak disease, though it cannot be stated definitely that such leaf markings are due in every case to infection with *Zea Virus 2*.

Dactyloctenium aegyptiacum Willd.

Tragus racemosus Scop.

Setaria verticillata Beauv.

Rottboellia exaltata L.f.

Cymbopogon citratus Stapf.

Diplachne eleusine Nees.

Euchlaena mexicana Schrad.

Panicum miliaceum L.

Avena sativa L.

Eleusine coracana Gaertn.

Imperata arundinacea Cyr.

Digitaria eriantha Stend.

D. horizontalis Willd.

D. marginata Link.

D. ternata Stapf.

Paspalum Scrobiculatum L.

Urochloa helopus Stapf.

Eragrostis valida Stent.

E. aspera Nees.

E. ciliaris Link.

Eragrostis sp., near to *E. porosa* Nees.

Eleusine indica Gaertn.

Geographical Distribution. The streak disease of maize caused by *Zea Virus 2* was first recognised at the end of the last century as an important factor limiting maize production in Natal. Since then the disease has been recorded from many parts of Africa, even as far north as Egypt.

Control. Comparatively few of the usual means of virus disease control are available in this instance. No naturally immune varieties of maize are known, nor is there any likelihood that any kind of artificial immunisation could be effective. No method of direct attack upon the insect vector offers any prospect of success. Owing, however, to the limited host range of the streak virus, the attack upon the virus during its carry-over stage between one crop and the next has favourable prospects. On the Natal coast this worked effectively, for, if no maize was grown during the winter in a district, the early planted summer crop usually escaped severe infection, even though infective insects might survive the winter in small numbers. On the other hand, if a succession of maize crops was raised through the year, very few plants ever escaped infection at any season. It seems, therefore, as if the best line of control lies in the extension of the period between crops to its maximum length. The destruction of any volunteer plants that may grow after the crop is harvested is important. The more nearly the sowings in a district are made at the same time, the less they should be subject to damage by streak. If a succession of sowings over a long period are made in a district, the conditions are ideal for a heavy infection of the later-sown crops (56).

ZEA VIRUS 3. Storey

Synonym. *Maize Mottle Virus*, Storey, 1937.

The Virus and its Transmission. This virus has been recently recorded for the first time by Storey (56A). It is not sap-transmissible, and the vectors are the same three species of leaf-hoppers as transmit *Zea Virus 2* (maize streak virus), i.e., *Cicadulina (Balclutha) mbila* Naude, *C. zea* and *C. nicholsi*. Much the same relationship exists between this virus and the *Cicadulina*

spp. as exists between these insects and *Zea Virus 2*. The active races of leafhoppers transmit *Zea Virus 3*, while individuals of the inactive race usually fail to transmit, although rare exceptions have been encountered.

Disease caused by *Zea Virus 3*

Graminaceæ

Zea mays L. Corn, maize. *Mottling disease* (see Fig. 77, C). An affected maize seedling under greenhouse conditions shows a transitory mottling of the young unfolding leaf. The effect is one of a rather diffuse blotching of normal green tissue upon a pale green background, the darker areas tending to follow the main veins. The diffuse nature of the pattern differentiates it from a typical mosaic, and in particular from the pattern produced by *Saccharum Virus 1* (sugar cane mosaic virus). The mottling is evident only during the early phase following infection. Seedlings in the greenhouse inoculated when the first leaf is just mature develop the first signs in their youngest leaves in about seven to fourteen days. As this leaf matures all symptoms fade and it assumes a uniform green. The next few leaves formed may show a similar pattern during their early growth; but after a time the plant produces young leaves devoid of mottling at any stage. Such a plant cannot be distinguished by inspection from a healthy one.

The young mottled leaves are not as rigid as those of a healthy plant and may fail to support themselves in the normal nearly upright position.

Geographical Distribution. *Zea Virus 3* has only been recorded from Tanga, Tanganyika Territory, East Africa.

Differentiation of the Diseases in Maize caused by *Zea Viruses 1 and 2*

The yellow areas of maize leaves affected by stripe (*Zea Virus 1*) tend to be longer without a break than those of streak (*Zea Virus 2*), and they may fuse together laterally to give broad, almost uniform yellow bands. The symptoms are, however, very variable and some types of stripe disease so closely resemble streak disease that differentiation may be very uncertain even by one well acquainted with both diseases. In such instances insect transmission experiments alone can give a certain diagnosis (56).

**Differentiation of the Diseases in Sugar Cane caused by
Saccharum Virus 1 and *Zea Virus 2***

While the pale areas in streak disease (*Zea Virus 2*) are long, narrow and straight and disposed along the veins of the leaf, those in mosaic (*Saccharum Virus 1*) are of irregular shape, and always spread unevenly laterally over several veins. While the markings of streak might be made, as it were, with a pen, those of mosaic would require brushwork. When the leaves are held up to the light, a pronounced difference of the contrast between pale and dark areas is observable in leaves affected with the two diseases. The light areas in the mosaic leaf (*Saccharum Virus 1*) are only a pale shade of green or yellow, while those of streak (*Zea Virus 2*) are quite devoid of green matter and are almost transparent (see Figs. 75, 77 and 78) (49).

TRITICUM VIRUS 1. McKinney

Synonyms. *Wheat Mosaic Virus*, McKinney, 1925; *Wheat Rosette Virus*, McKinney, 1928.

The Virus and its Transmission. There is no information available on the properties or insect vectors of this virus. It seems, however, to be sap-inoculable and it is not carried in the seed. According to Dufrenoy (18) the virus is not filterable. An unusual feature of this disease is the fact that the virus seems to be soil transmitted and it can remain infective in the soil for so long as six years (compare *Nicotiana Virus 11*, p. 266). The exact mechanism of this soil infection does not appear to be known, but Webb (63) states that infection of wheat seedlings may and does occur through either the roots or the crown or both. The disease develops when the infested soil is either below, above or lateral to the seeds and at a considerable distance from them. When the infested soil occurs only below the grain, from 28 to 55 per cent of the plants show symptoms of rosette; when the soil occurs only above the grain, 22 to 39 per cent; and when both below and above, usually 70 to 95 per cent. The tissues of and near the crown appear highly susceptible. Five inches below the seeds was the greatest distance at which infested soil caused the rosette expression. Mottling develops with infested soil at a depth of 10·5 inches below the seeds.

Diseases caused by *Triticum Virus 1*

Graminaceæ

Triticum sativum or *vulgare*. Winter wheat. *Secale cereale*. Rye. **Rosette.** The mosaic pattern caused by this virus on the leaves of wheat and rye is much smaller and less conspicuous than the mosaic pattern on the leaves of sugar cane, probably because of the smaller size of the leaves of wheat and rye. The mottling is rather rare on the wheat plants in late autumn, but develops more abundantly on the new leaves in early spring. The mottling consists of irregular streaks, which vary in length and width and tend to follow the direction of the long axis of the leaf. In some cases mottled leaves show a light green pattern on the normal green background. Frequently, however, the greater proportion of the infected leaves are of the lighter green colour, making it appear as if there was a dark green pattern on a light green background. In certain varieties the leaves frequently develop light yellow patterns or irregular strips. It is not uncommon to find mosaic mottling also on the leaf sheaths and glumes. In addition to the mosaic mottling, the virus causes stunting or dwarfing and excessive proliferation in certain varieties. This condition, which used to be described as "rosette," causes a field to take on a spotted or patchy appearance on account of the various sizes of the areas which contain the stunted plants. This rosette condition is especially severe in certain American varieties of wheat, such as Harvest Queen.

The leaves of rosetted plants eventually become dark green in colour, thus masking the mosaic mottling. The cell-inclusions typical of the disease, however, have always been found in such plants. When such plants send out new tillers, mosaic has been found to occur on the new leaves of these tillers before the dark green colour develops (34).

Histopathology. Intracellular inclusions, or X-bodies, are present in the cells of the diseased wheat and rye. The inclusions are quite typical of this kind of reaction. They are vacuolate, usually rounded and often in close association with the cell nucleus (36).

Host Range. *Triticum Virus 1* is transmissible to all members of the *Hordeæ*, and the following species have been found susceptible to infection: *Triticum vulgare*, *T. compactum*, *T. turgidum*, *T. durum*, *T. dicoccum*, *T. spelta*, *T. polonicum*, *T. monococcum*, *Hordeum sativum* and *Secale cereale* (85).

Geographical Distribution. The disease is widespread in the

United States of America ; it has been found in Madison, Mason, Logan and Cass Counties in Illinois, in La Porte and Porter Counties in Indiana, and Rosslyn, Virginia.

Control. The best method of control for the mosaic and rosette diseases of wheat caused by *Triticum Virus 1* is the use of resistant varieties. Infested fields, in the United States, should not be sown with the following varieties : Harvest Queen, Missouri Bluestem, Nigger, Penquite, Brunswick, or certain selections of Fultz, Indiana Swamp, and Illini Chief. These varieties are all very susceptible to both the mosaic and rosette forms of the disease. Although the Currell variety does not develop rosette, it appears to be very susceptible to mosaic and it should not be sown on infested soil. Most wheat varieties seem to be susceptible to the mosaic form of the disease, but in many cases the disease does not seem to be very severe. Certain selections from the variety Harvest Queen seem to be resistant to the virus (34).

Strains of *Triticum Virus 1*

It appears from the work of McKinney (85) that the virus occurs in strains of the "green" and "yellow" types similar to those strains associated with *Nicotiana Virus 1* and *Cucumis Virus 1*. Later work, however, may show that more than one virus is concerned with the mosaic disease of winter wheat and rye.

TRITICUM VIRUS 1A. *Yellow mosaic*

The yellow type of mosaic produces a bright yellow mottling and causes the plants to become dwarfed late in the spring and the seed to be shrivelled and practically valueless.

ORYZA VIRUS 1. Fukushi

Synonyms. *Rice Stunt Disease Virus*; *Rice Dwarf Disease Virus*.

The Virus and its Transmission. There is no information on the physical and other properties of this virus, since it is not sap-transmissible. It is not carried in the seed or in the soil. The specific insect vector is the leafhopper, *Nephotettix apicalis*, var. *cincticeps* (see p. 487), and there are several interesting points in the relationship of virus and insect vector.

All individuals of the leafhopper are not capable of transmitting the virus and certain individuals fail to transmit the virus even when they have been hatched and reared upon diseased plants. It is not known whether such insects are analogous to the "inactive" races of *C. mbila*, the leafhopper vector of *Zea Virus 2* (see p. 442). The majority of the progeny of an infective female are capable of acting as vectors, and an insect, once infective, usually retains the virus for the rest of its life.

Sometimes a period of five minutes is sufficient for a single leafhopper to feed upon a healthy plant in order to transmit the disease, and a feeding period of thirty minutes is generally long enough for infection of healthy plants. Viruliferous leafhoppers occasionally lose their infectivity even under favourable conditions, and viruliferous insects do not necessarily infect every healthy plant on which they feed. A minimum period of three days seems necessary for a leafhopper to feed on a diseased plant in order to pick up the virus.

According to Fukushi (16) this virus is transmitted through the eggs of infective leafhoppers. The progeny from infective female leafhoppers are either viruliferous or free from the virus, whereas the progeny from the crosses between uninfective females and infective males are entirely non-viruliferous. It appears that all the ova in the ovaries of an infective leafhopper are not always affected by the virus. A period of one to fourteen days must elapse before most of the infective nymphs become capable of producing infections in healthy plants, although certain individuals may be infective immediately after emergence from the eggs. Fukushi suggests one or two alternative explanations for this phenomenon. (1) The newly emerged nymphs may not be able at first to transfer sufficient virus to cause infection. This theory, of course, involves multiplication of the virus in the insect. (2) Some developmental changes in the virus may occur within the insect's body before it is fully infective. (3) It may be necessary for the virus to migrate from another part of the insect's body to the salivary glands and the anterior portion of the alimentary canal. Most of these infective nymphs retain their infectivity during all the nymphal stages and through the entire adult life without access to a source of virus.

According to some more recent work of Fukushi (18), the virus can be passed on to the third generation of leafhoppers, and this is considered to be strong presumptive evidence that the virus is actually multiplying inside the insect.



A



B

C

D



E

Diseases caused by *Oryza Virus 1*

Graminaceæ

Oryza sativa. The rice plant. *Dwarf (or stunt) disease* (see Fig. 78, A and D). The first visible sign of the disease manifests itself as yellowish-white specks along the veins of newly unfolded leaves. These specks, which develop before the leaves unfold, are yellowish-green to yellow when viewed by diffused light. On holding up to the light, these specks become distinct, being yellowish-white to white in colour. The specks elongate and spread out along the leaf parallel to the mid-rib, forming fine interrupted streaks. These range from mere dots to an area several millimetres in length and from 0·2 to 1 mm. in width. The succeeding leaves invariably show the white specks, while the lower, previously formed leaves exhibit no signs of the disease. On the leaf which shows the first visible symptoms the specks may be confined to the lower part of the leaf blade or to only one side of the mid-rib near the base of the leaf. On the succeeding leaves more conspicuous specks develop in abundance and connect with each other, forming almost continuous streaks along the veins. Thus the symptoms of the disease become most pronounced about the middle of July.

Growth subsequent to infection being much arrested, the diseased plant becomes remarkably stunted, with the internodes shortened, while numerous diminutive tillers develop, producing a rosette appearance. Affected plants tend to develop a dark green colour in the foliage and their roots are arrested in growth, only small roots, extending horizontally, developing. Plants infected in early stages of growth become severely stunted, being at most only 10 inches high. They produce a few worthless panicles or none at all.

Histopathology. Studies of sections of diseased leaves show chlorotic modifications in the mesophyll cells adjacent to some of

FIG. 78.

A-D. *Oryza Virus 1* (causing dwarf disease of rice).

- A. Healthy (*left*) and diseased rice plants.
- B. Leaf of diseased plant of *Echinochloa Crus-galli* var. *edulis*.
- C. Leaf of diseased plant of *Panicum miliaceum*.
- D. Leaf of diseased rice plant.
- E. Leaf of maize plant infected with *Zea Virus 2* (Streak); photographed by transmitted light.

(A-D, after Fukushi; E, after Storey & McClean.)

the vascular bundles. In sections mounted in water, the chlorotic tissues are lighter in colour or nearly colourless, the chloroplasts in these cells being light coloured and smaller in size and number. In cells where the chloroplasts have been most disintegrated, intracellular inclusions are usually present. The X-bodies are more abundant in the mesophyll cells, but it is not unusual to find them in the epidermal cells and they are frequently in close association with the cell nucleus. The bodies vary considerably in shape and size, the most usual forms are round or oval, but amoeboid or irregular shapes are not uncommon. In size they range from 3 to 10 μ in length and 2.5 to 8.5 μ in width. They are vacuolate and are characteristic of this type of pathological response.

Host Range. The following grasses are susceptible to infection with this virus : *Panicum miliaceum* L., *Echinochloa crusgalli* Beauv., subsp. *colona* Honda, var. *edulis* Honda, *Alopecurus fulvus* L. and *Poa pratensis* L. Rye, wheat and oats are only slightly susceptible to the virus, while maize is apparently immune (see Fig. 78, B and C).

The symptoms on the susceptible grasses closely resemble the disease on the rice plant, being characterised by streaking and spotting of the leaves and stunting of the whole plant. Similar intracellular inclusions are also present (17).

Geographical Distribution. *Oryza Virus 1* appears to be confined to Japan. It is prevalent in the middle and southern prefectures, but is scarce or absent in the north.

Control. There is very little information on control methods for *Oryza Virus 1*. There is some evidence that the virus overwinters in the insect rather than in some wild host plant, so that any methods to destroy the leafhopper in its hibernating quarters would be useful. At present nothing seems to be known of resistant varieties of rice.

ORYZA VIRUS 2. Kuribayashi

Synonym. *Rice Stripe Disease Virus*, Kuribayashi, 1981.

The Virus and the Disease caused by it. The stripe disease of rice was first described from Japan in 1981 (26). It is somewhat similar to the disease caused in rice by *Oryza Virus 1* in the stunting produced, but differs in the development of yellow stripes along the leaves. The virus is transmitted by a different species of leafhopper, *Delphacodes striatella* Fall. (see p. 492) (25).

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CHAPTER VIII

THE INSECTS, ETC., CONCERNED IN THE TRANSMISSION OF PLANT VIRUSES

In the ensuing chapter an account is given of the insects, etc., which are concerned in the transmission of the plant viruses dealt with elsewhere in this book. A detailed description of each insect is given, together with an account of its life history and habits, so far as these are known, food plants, viruses transmitted and geographical distribution. Whenever possible an illustration of the insect is also given.

Probably the systematic entomologist will not be satisfied in every case with the nomenclature adopted, but the synonymy of insects, and especially aphides, is a vexed question, and the writer has done his best to follow the latest ruling on the subject. The following points which have arisen since this chapter was written will help to illustrate the difficulties. According to Lambers (29) *Macrosiphum gei* is a differert aphis from *M. solanifolii*, although, following Theobald's ruling, the writer has treated the names in this book as being synonymous. If two different species are concerned, however, it will perhaps explain, as Floyd Smith has suggested (46), the apparent difference in the virus-transmitting power in America and England of what has hitherto been considered only one species. Again, according to Steele (49), the thrips vector of *Lycopersicum Virus 3* (tomato spotted wilt virus) in Australia is not *Frankliniella insularis*, but a new and undescribed species of thrips. *Cicadula sexnotata* Fall., long known as the vector of *Callistephus Virus 1* (aster yellows virus), is now said to be a European species, while the name of the vector, which is only found in North America, should be *Cicadula divisa* Uhler, and this has recently been changed to *Macrosteles divisus* Uhler.

If the types of insects concerned in plant virus transmission be examined, it will be seen that, although there are several leafhopper vectors, the aphis vectors predominate and the aphis *Myzus persicae* is now associated with no less than twenty-one plant viruses. Only one capsid bug has up to the present been

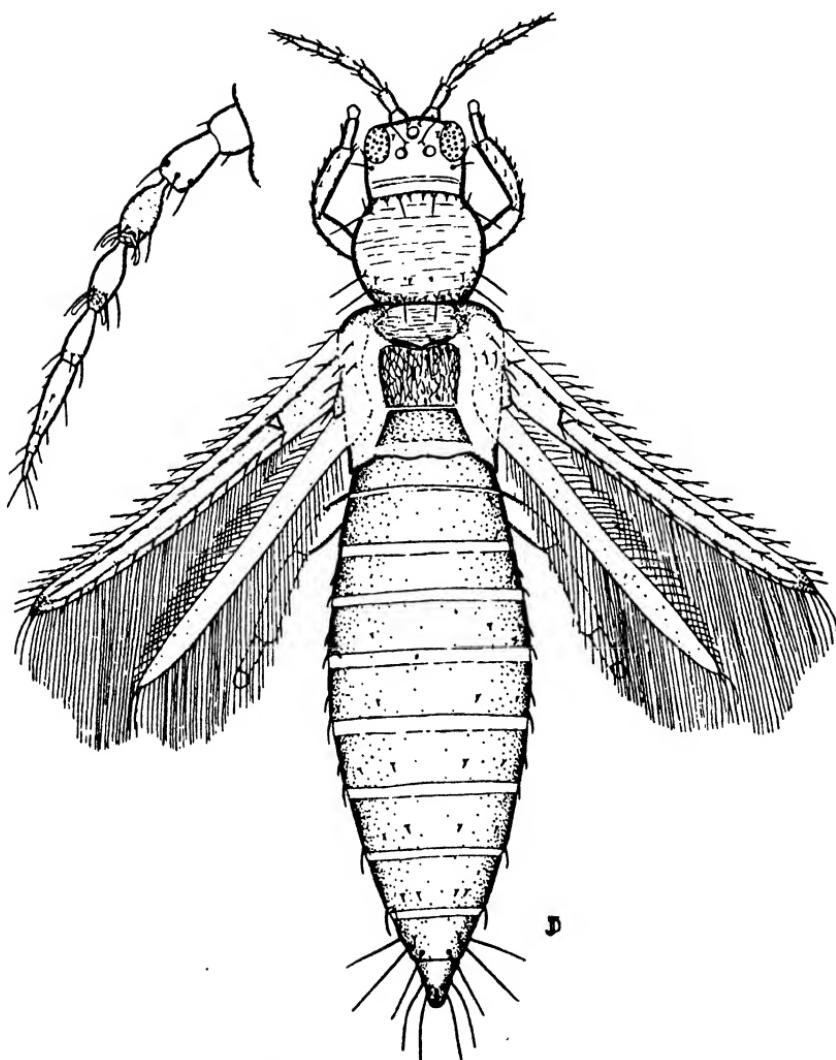


FIG. 79. *Frankliniella insularis* Frank. Adult female, black carnation thrips.

Nat. size, length 1.3 mm.; width of mesothorax 0.25 mm. (After Davidson and Bald.)

identified with virus transmission, this is *Lygus pratensis*, which transmits a virus affecting cruciferous plants in Germany (25).

THYSANOPTERA

Frankliniella insularis Franklin. The black carnation thrips (see Fig. 79).

DESCRIPTION

Egg. Whitish, ovate, shaped somewhat like a kidney bean, being about 0.7 mm. long by 0.3 mm. broad.

The Female. Length, 1.3 mm.; width of mesothorax, 0.25 mm. Colour, deep brown to blackish-brown; thorax and abdomen somewhat lighter brown than head; antennæ with two proximal segments dark brown, middle segments lighter, grading into dirty brown on distal segments; eyes dark brown, faintly tinged with red; ocelli brownish-yellow with crescent area darker; wings paler than body, fringe brown; legs with coxae and femora brown; tibiae lighter, especially fore tibiae; tarsi yellowish-brown. **Head**, length 110 μ , width 165 μ , cheeks straight, faintly narrowing proximally, a transverse ridge on dorsal surface about one-fifth from posterior margin; **eyes** about 54 μ antero-posteriorly and 22 μ transversely; ocelli well separated; ocellar bristles 44 μ , situated at anterior ends of posterior ocelli; postocular bristles about 34 μ , two small bristles situated in front of anterior ocellus near median line, a small bristle on each side of anterior ocellus towards inner margin of eyes; on ventral side mouth-cone extends to coxa I., length from base of antennæ to tip of mouth-cone about 208 μ , two bristles on each side behind base of antennæ, the pair nearer the median line being the longer, about 35 μ ; a small bristle on each side near postero-internal margin of eyes; two bristles about 25 μ on each side of median line near posterior margin of head; **antennæ** about 0.26 mm. long; relative lengths of segments, 17.6, 33, 50.6, 44, 33, 50, 11, 17.6; segments bear prominent bristles about 26 μ long, especially on distal part of segments; forked sense-organ near distal end of segments III. and IV.

Prothorax. 143 μ long, 220 μ wide, sides rounded; on dorsum a long bristle 66 μ near each antero-lateral angle directed posteriorly, also a small one 18 μ directed anteriorly; six very small bristles forming a row along anterior border; two long bristles about 70 μ near each post-lateral angle; two shorter bristles 38 μ , one on each side of median line near posterior border, and a row of about twelve small bristles along posterior border; a few small bristles scattered over posterior area of pronotum.

Pterothorax. Mesoscutum with two short bristles near post-lateral angles, and one or two other short bristles along posterior margin; metascutum I. with four prominent bristles along anterior border, close together, the two nearer median line about 55 μ long, and the other two 80 μ ; on ventral side a row of three bristles extending along inner post-lateral border of mesothorax, and a conspicuous bristle near

post-lateral angle of metathorax ; a few smaller bristles scattered over venter of pterothorax.

Wings reach to segment VII. ; anterior margin of forewings bears about 20μ stout bristles 66μ long, interspersed with others not so stout ; anterior vein bears about seventeen and posterior vein about fourteen short stout bristles. Legs bear rows of short stout bristles, two situated on inner face of each tibia at the distal end stouter than the remainder ; the spines on inner face of tibiae of posterior legs stouter than on the others.

Abdomen. Pleural plates connecting tergites and sternites are denticulate on posterior border, a stout spine at post-lateral angle of each segment, except segment I., also a spine about the middle of lateral margin of segments ; bristles on segment IX., four conspicuous bristles on tergum, two outer bristles about 120μ , and two nearer median line about 103μ ; on segment X. also four conspicuous bristles, the pair nearer the median line about 126μ , and the outer pair about 116μ ; a few smaller bristles on each segment ; on the venter sternites bear a prominent row of six bristles about 33μ long on posterior borders of segments II. to VII. ; on segment IX. two conspicuous stout lateral bristles about 88μ , and on segment X. about 66μ ; a row of fine bristles on each side of median line over segments VIII. to X.

The Male. Smaller and narrower than the female ; length about 1 mm., width of thorax 0.2 mm. *Colour*, general colour lighter than in female ; on venter of abdominal segments III. to VII. a distinct sole-shaped, light coloured area. *Head*, length 110μ , width 132μ ; *antennae*, about 240μ , relative length of segments, 20, 33, 48, 39, 33, 44, 9, 13. *Wings* reach to abdominal segment VII. *Abdomen*, on segment IX., two long bristles each about 72μ , two near median line quite short, 9μ , and two on post-lateral angles each 66μ ; on segment X. two bristles on post-lateral angles each 66μ ; on venter of segment IX., two lateral bristles each 44μ , and two on post-lateral angles as in the other segments ; on segment X. one bristle at each post-lateral angle each 66μ long (10).

Food Plants. This thrips has a very wide host range ; in South Australia it has been collected from more than forty different food plants as follows : *Ageratum*, *Agapanthus*, *Bignonia*, *Cichorium*, *Lonicera*, *Valeriana*, *Delphinium*, geranium, *Jacaranda*, larkspur, melon, pansy, stock, carnation, verbena, *Phaseolus*, *Malva parviflora*, *Oxalis cernua*, roses, jasmine, sweet peas, *Coreopsis*, cornflower, cosmos, foxglove, gladiolus, oleander, poppy, *Zinnia*, *Antirrhinum*, fuchsia, hollyhock, lucerne, *Opuntia*, *Petunia*, potato, strawberry, ulex, cucumber, dahlia, Easter lily, tomato.

Life History and Habits

So far as the tomato plant under glass is concerned, and it is the virus transmitted to and from this plant which gives this

thrips its economic importance, the eggs are laid in the tissues of the leaf, and the larvae feed on the leaves or in the flowers; when mature they leave the plant and pupate in the *débris* on the surface of the soil or penetrate into the upper layers of the soil. Sometimes they pupate on the plant in rolled leaves, depressions in the stem or other similar situations; this habit has been observed on tomato plants growing in the open during the summer. In carnation flowers eggs are laid in the tissues of the sepals and petals. The duration of the egg stage varies from nine to fourteen days at a mean daily temperature of 64·5° F. The duration of the larval stages varies from nine to thirteen days at a mean daily temperature of 66·6° F. to 65·7° F. The duration of the pupal stage varies from seven to fourteen days at a mean daily temperature of 72° to 66° F. The complete life cycle under glasshouse conditions occupies thirty-six to thirty-nine days, when the mean temperature of the period is about 66° F.

Ecology. Under glasshouse conditions with mean temperatures below about 62° F. the developmental period for the life cycle of the insect becomes rapidly extended and the mean monthly temperatures of May to October explain the scarcity of *Frankliniella insularis* during these months in the Adelaide district. As the insects have been readily reared in the glasshouse at all periods of the year, the factor which limits its distribution and multiplication in the open is evidently temperature (10).

Virus Transmitted by Frankliniella insularis
Lycopersicum Virus 3, causing spotted wilt disease of tomatoes.

Geographical Distribution. The chief distribution of *Frankliniella insularis* appears to be the region of Central America. It is common in the Adelaide area, South Australia, during the warmer period of the year (approximately November to April), and rare or absent during the remaining months. It does not seem to occur in Europe.

THYSANOPTERA (*continued*)

Thrips tabaci Lindeman. Onion thrips.

DESCRIPTION

Adult. Recognisable by the rather slender antennæ, the short body-bristles, the generally light coloured antennal segment I., but

especially by the size of the body and the presence, generally speaking, of four distal bristles on the main vein of the forewing.

Female. Body-colour pale yellow, body dorsally sometimes more or less dark grey coloured. Body-bristles mostly brownish or blackish, now and then rather pale. Antennal segment I. whitish-yellow or hyaline, rarely pale grey, the remaining segments grey, III. yellowish, generally dark at the end, IV. and V. pale at base, as also (more rarely) VI. The first segment is as a rule the palest. Legs yellow, femur and tibia often more or less dark grey. Wings pale yellow, sometimes pale yellow-grey.

Head broader than long, at the eyes as broad or only slightly narrower than at the strongly arched, posteriorly contracted genae; these indistinctly serrated; vertex wrinkled. Body-bristles small, yet well developed, in normal positions. Eye facets not protuberant. Mouth-cone rather less slender than in *flavus*; of the maxillary palp segments, as in the above species, II. is the shortest, III. the longest, but only slightly longer than I. Antennae 225 to at most 250 μ long, slender, the segments slightly rounded laterally, I. rather broader than long, II. about as long as V., III. rather longer than IV., V. of medium length, VI. about as long as III., at most 2·8 to 2·9 times as long as the short, in side-view somewhat truncated VII. Prothorax almost 1·4 times as broad as long, anteriorly somewhat contracted, 1·3 to 1·4 times as broad as, and rather longer than, the head. Bristles on anterior border insignificant. Bristles on posterior angles comparatively very short, measuring 35 to 43 μ , the outer ones are somewhat longer than the inner ones. The hind border between the angles with three small bristles on each side, of which the innermost pair are about as long as the longer pair of angle bristles. Pterothorax 1·25 to 1·3 times as broad as prothorax, only slightly longer than broad. Wings with well-developed veins, the anterior ones 0·65 to 0·68 mm. in length. Costa with twenty-seven to thirty, less often only twenty-four to twenty-six, bristles of medium length; main vein with seven basal and as a rule four (two and two or two and one and one), rarely with five to seven, exceptionally with only three distal bristles on both sides; parallel vein with at most fifteen to seventeen, more rarely eleven to thirteen, bristles.

Abdomen fairly broad. Segment VIII. dorsally at hind border with long complete comb of bristles. Posteriorly situated bristles of medium length, on the ninth tergite the dorsal pair measures 38 to 46 μ , of the bristles on the hind border the inner ones measure 62 to 78 μ , the outer ones 86 to 95 μ . The bristles on segment X. (dorsal) are 70 to 75 μ long. Ovipositor 0·17 to 0·18 mm. long. Body length, 0·8 to 0·9 mm. (extending to 1·2 mm.).

Male. Yellow, thorax deep yellow. Antennal segment I. hyaline, the remaining segments darker, III. to VI. often pale at base. Forewings as in female, with four or frequently three distal bristles on the main vein, rarely one or two. Antennae as in female. Body much narrower. The dorsal transverse row of bristles on tergite IX. as in *montivagus*,¹ except that the bristles are less closely set, 27 to 30 μ long. The lateral elements of the penis are rather short. Sternites III. to VII.

¹ The ninth segment bears dorsally six bristles, of which the four long middle ones (82 to 88 μ) are set very closely.

each with a transverse elliptic cavity ; the breadths of the cavities on the third, fourth, fifth, sixth and seventh segments measure typically 35, 38, 41, 30 and 22 μ respectively. Body length, 0.7 to 0.75 mm. (extending to 0.9 mm.).

Egg. Elliptical, curved, pale whitish, 0.26 mm. long and 0.12 mm. broad.

Larva. First instar. In this phase the larva is whitish to pale yellow, the eyes are reddish. Undoubtedly very similar to the larvae of the other yellow species, but may be distinguished from many of these by the relatively shorter bristles.

In the second instar the larva is bright yellow, like the larva of *nigropilosus*. " Legs vaguely grey, antennae pale grey, segment III. with hyaline ring near the tip. Segment IX. with narrow grey hind border. Antennæ shorter than in above species (*T. flavus*, *nigropilosus*, *alni*), with shorter and more delicate bristles. The last segment is about 2.4 times as long as broad. Bristles on the posterior angles of the prothorax much shorter, 19 to 23 μ long, the lateral bristles on tergite IX. 38 to 42 μ long. Shape of abdomen similar to that of above species, though somewhat slenderer. Setacomb on hind border of tergite IX. scarcely noticeable, extremely delicate, much finer than in the other species."

Measurements. Antennal segments from II. onwards : 22 (19), 35 (22), 41 (19), 8 (11), 16 to 17 (7) μ . Breadth of mesothorax : 187 to 204 μ . Body length of full grown larva : 0.78 to 0.9 mm.

Pupa. According to Quaintance, about 0.7 mm. long. Breadth of mesothorax, 0.15 mm. Yellowish to almost colourless, eyes reddish. Wing-sheaths reach to abdominal segment VIII. Tip of abdomen with transparent curved spines. A more exact description has not as yet been published.

Occurrence. An uncommonly prevalent, sometimes injurious, species, causing serious damage to onions and cabbages, especially in North America. In Roumania and other places it is notorious as a pest of tobacco. According to Knechtel it causes leaf-roll of *Nicotiana* seedlings. Ludwig records it as a pest of *Helleborus foetidus*, etc. (39).

Diagnostic Characters of *Thrips tabaci* (see Fig. 80).

- Female.** (a) Ocellar crescents grey, colourless by reflected light.
- (b) Only one (posterior) pair of seta-pits present upon ninth abdominal tergite.
- (c) Complete fringe of seta across hind margin of eighth abdominal tergite ; fine and close-placed at regular intervals.
- (d) Setæ at lateral posterior margins of prothorax short.
- (e) Three setæ with anteriorly placed seta-pit along lateral margin of second abdominal tergite.
- (f) First abdominal sternite devoid of setæ.
- (g) Epipleurites and sternites of abdominal segments II. to VII. inclusive, devoid of *intermarginal* setæ.

Male. Characters (a), (b), (d), (e), (f), (g) as in female.
 Light depressed areas present only on abdominal sternites III., IV. and V., entirely absent on VI. and VII. These are transversely long

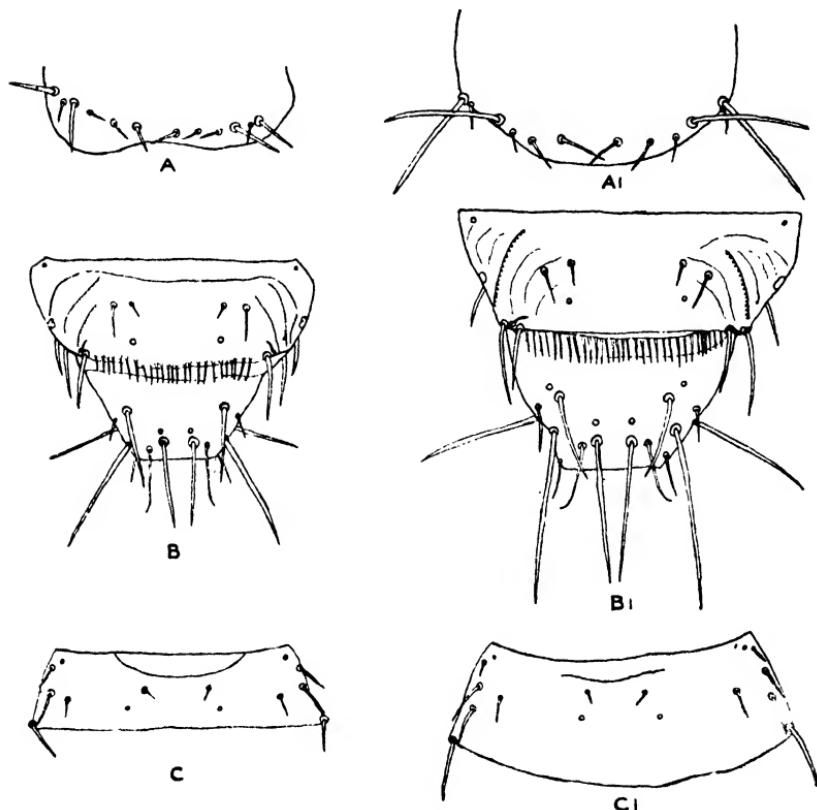


FIG. 80. Diagnostic characters of *Thrips tabaci* Lind., female imago (A-C) and of *Thrips flavus* Schrank, (A1-C1). $\times 350$.

A and A1. Setae at posterior margin of prothorax.
 B and B1. Chaetotaxy of eighth and ninth abdominal tergites.
 C and C1. Chaetotaxy of second abdominal tergite.
 In A1, note greater length of setae at lateral corner.
 In B, note absence of anterior pair of seta spikes on ninth abdominal tergite.
 In C1, note presence of four setae along lateral margins (only three in C).
 (After Speyer.)

and very narrow; ninth abdominal tergite with two anterior and a row of four posterior setae.

Antennæ. Joint I. colourless, II. yellow, III. peduncle colourless with a brownish-yellow transverse band at apex. Proximal two-thirds colourless, distal one-third brownish-yellow; V. peduncle grey,

proximal half yellow, distal half brownish-yellow ; VI. proximal half yellow, distal half brownish-yellow ; VII. brownish-yellow (47).

Food Plants. Depending on the plant, the locality and other factors, both adults and nymphs may feed amongst the flowers, green fruit, the succulent, more sheltered parts of the stem, the leaf-buds, the tender surface of leaves and rarely on the upper surface of shaded leaves with a delicate cuticle. The insect may be found on a great variety of wild and cultivated flowering plants. Morison records it from more than seventy-four different species of plants. He states that its chief host plants in the open in Great Britain are certain Compositæ and Papilionaceæ. During July to September, in north-east Scotland various small isolated plants of *Achillea millefolium*, *Matricaria inodora* L. and *Spergularia marina* may be hosts to large colonies of *tabaci* of both sexes. The insect is also a pest of onion, pineapple and tobacco in various countries. In glasshouses in the British Isles it feeds on a great variety of different plants and breeds freely upon the foliage of cucumber and cineraria, and within the flowers of carnation, arum and cyclamen. Adults frequently collect together in the flowers of chrysanthemum (47).

Life History and Habits

In north-east Scotland, dark ovigerous females appear in April after hibernating, probably as adults. During warm weather and in bright sunlight they may jump to a height of 1 cm. and a distance of several centimetres, but they rarely attempt to fly. Apparently they oviposit in the more succulent plants during April and May, and their nymphs appear in June and become adult in July, when males appear amongst the progeny. This first generation gives rise to a second generation, including males, maturing in September and October. There is an overlapping between the first and second generations during July and August, probably owing to the different dates of emergence of the hibernated females and to the length of the oviposition periods. The males disappear during October and the females seem to hibernate till the following year (Morison). Out of doors, in England, the thrips does not become common upon cultivated or wild plants until July, and larvæ have been taken from flowers of yarrow at the beginning of November. In autumn the adults congregate with other species in the flowers of rose and of various other cultivated plants, and sometimes in those of the dandelion. Reproduction in this species is almost entirely parthenogenetic in

England; the only authentic record of the occurrence of males comes from Scotland by Morison, who collected them from *Cochlearia officinalis* and *Senecio jacobaea* (Speyer).

MacGill (30) gives the duration of the life cycle upon the cotton plant grown under glass as follows: Egg, eight days; larval stages, ten to fourteen days; prepupa, one to two days; pupa, four to seven days; total about four weeks. Seven parthenogenetic generations are recorded by her during the summer. The female has been observed to breed throughout the year upon the flowers of arum, grown under glass, without the appearance of the male sex upon any occasion.

Ecology. In studies upon the relation between variations in temperature and the life cycle of *T. tabaci*, MacGill (30) comes to the following conclusions. The exposure of larvæ to temperatures of 38°, 31°, 8° and -4° C. for periods varying from one to ninety-six hours has a marked effect on the survival of the insects. At 100 per cent relative humidity exposure to 38° and 31° C. gives a higher rate of mortality than exposure to 8° and -4° C.; at 82 per cent relative humidity 38° and -4° C. are the least favourable temperatures, and at 75 per cent relative humidity -4° C. is the most unsuitable for the survival of the insects. In some conditions more larvæ survive after a long exposure than after a shorter one, which suggests that at certain points in their life cycle the insects are particularly sensitive to changes in their environmental conditions. From these experiments it does not appear that relative humidity has a marked effect on the length of the life cycle. Exposure to low temperatures has a more constant effect on the length of the larval stage than exposure to high ones; exposure to -4° C. retards development for a longer time than the duration of the exposure, and exposure to 8° C. for a shorter time. The length of the life cycle varies with the absolute humidity of the atmosphere, and at vapour pressures below 15 mm. the conditions have a retarding influence on development, while those between 15 and 25 mm. accelerate development.

Viruses Transmitted by *Thrips tabaci* L.

Lycopersicum Virus 3, causing spotted wilt disease of tomatoes.
Ananas Virus 1, causing yellow spot disease of pineapples.

Geographical Distribution. The distribution of *Thrips tabaci* is almost world-wide; it is common in Europe generally, in many parts of North America, Australia, and Hawaii.

HEMIPTERA-HETEROPTERA

Capsidæ

Lygus pratensis Linn. The tarnished plant bug.

DESCRIPTION

Very variable in colour, being generally brown to reddish-brown or yellow. Head yellowish-brown, usually marked with three longitudinal lines brownish, black or reddish in colour, very variable in distinctness. Prothorax bronzy-brown, usually with four more or less distinct blackish spots in a row, one-third the distance from the front margin, sometimes so arranged as to give the prothorax the appearance of having four longitudinal dark stripes; the posterior angles of the prothorax are marked with a dark brown, black or red spot. Scutellum varies from brown to black and is usually marked with a heart-shaped or Y-shaped spot on the posterior half. Wings bronzy-brown, mottled with yellowish-brown and reddish. Antennæ dark brown, first segment and second segment except the tip usually lighter. Legs light yellowish-brown to dark reddish; under surface of insect dark in the centre with a lighter stripe on each side. There is a marginal band of brown in which is a submarginal row of yellowish spots, one to each segment. As a rule the male is smaller and darker than the female. The ovipositor, when at rest, lies nearly concealed in a groove on the under surface of the abdomen. Length, 5 to 6 mm. (7).

Egg. Varies slightly in length and diameter, average measurements being 1 mm. long and 0·24 mm. in diameter at the basal end. General outline flask-shaped and slightly curved, base somewhat enlarged and rounded; the apex is narrowed and rimmed, the margin of the rim being thickened and darker in colour; at first transparent, later becoming pale yellow (57).

Food Plants. *L. pratensis* has a very wide range of food plants. It attacks a variety of crops, fruit trees and ornamental plants. It has been recorded as a pest of potatoes, beans, beet, celery and Brassicæ. It occurs commonly on the stinging nettle, and in England it may be found in large numbers in September on the black nightshade (*Solanum nigrum*).

Life History and Habits

In New York the tarnished plant bug hibernates in the adult stage and becomes active again with the first warm days of spring. The adults may attack the opening buds of fruit trees like the apple, pear and plum, and are later found abundantly in grassy fields and on low-growing weeds. The eggs are usually deposited in the leaf-petiole or mid-rib. In Brassicæ the eggs may be found in the seed stalks, stems or leaves, especially of the turnip, *Brassica campestris*. They are also laid in the flower heads of certain species. In England and probably in Europe generally

there appear to be two generations a year, hibernation again being in the adult stage.

Virus Transmitted by *Lygus pratensis*

An unclassified virus affecting turnips and rape in Germany (see p. 21).

Geographical Distribution. *L. pratensis* is very widely distributed ; it occurs practically all over Europe and North America.

HEMIPTERA-HETEROPTERA Tingidæ

Piesma (Zosmenus) quadrata Fieb (see Fig. 81).

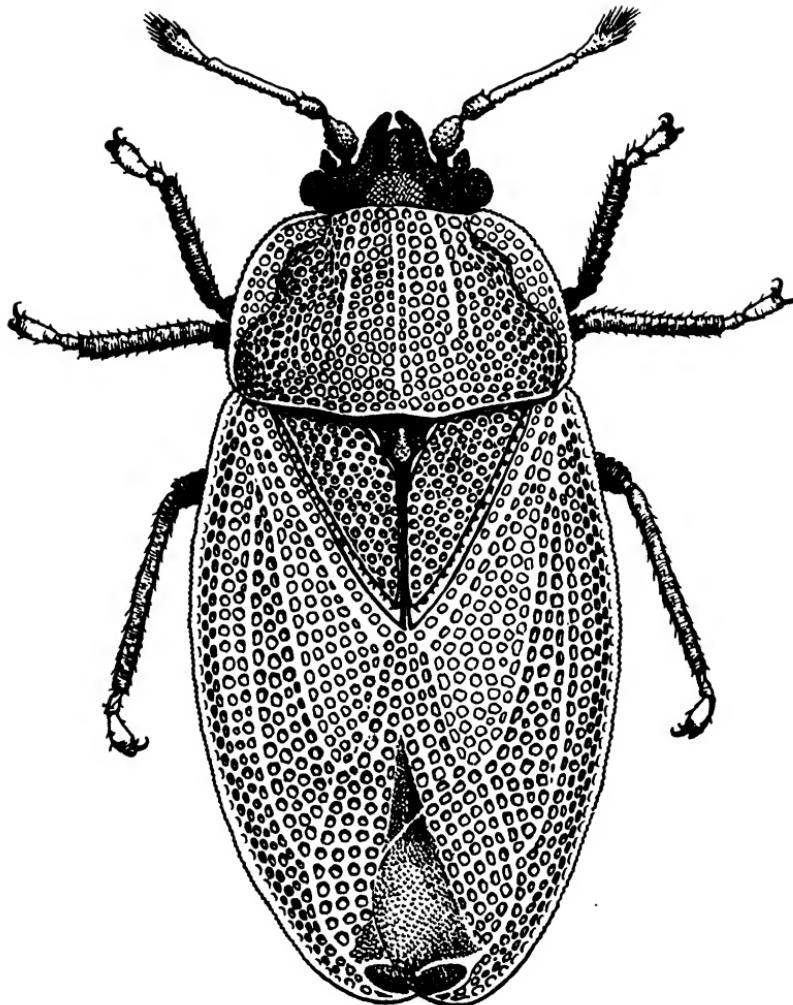
DESCRIPTION

Egg. 0·64 mm. long, 0·27 mm. thick (mean of fifty measurements), somewhat flask-shaped, yellow. Size and shape variable. In sections the egg is not circular, but slightly compressed laterally. The posterior pole is rounded, and normally corresponds to the posterior end of the developing embryo, and comes first out of the genital opening. The anterior pole, corresponding to the head-end of the embryo, is obliquely flattened and bears on its flat surface a ring of small protuberances. The longer and usually convex side of the egg corresponds to the dorsal side, the shorter and less curved (in extreme cases slightly concave) side, to the ventral side of the developing larva. Egg usually attached to its support by the dorsal side. Broad shallow furrows occur longitudinally in the integument, persisting even after the larva has emerged. The surface of the integument is not quite smooth, but is covered with very short stumpy transparent bristles, which occur in irregular six-sided formation. The integument is tough and can withstand considerable pressure. On the obliquely flattened end occur six, sometimes five, rarely four, cone-shaped tubes, in a ring ; the spiracles. The distal dark coloured ends of these breathing tubes appear to end in an opening which may be covered with a thin membrane. The egg colour varies during the course of embryonic development. Fresh-laid eggs are whitish to light yellow, becoming darker yellow ; later appear the red eyes and abdomen of the larva showing through. Just before eclosion, the egg is dark brown. The empty "shell" is light yellow and shows an iridescent light brown or violet colour by reflected light.

Larva. There are five instars, which during their development through four ecdyses increase in size from about 0·7 to 2·3 mm.

The body of the larva is boat-shaped, dorsally flattened, and deeply rounded ventrally. The segmentation is very distinct in all instars. Behind the large head (relatively very large in instar I.), occur three thoracic segments and nine abdominal segments.

The larva in its first instar is pale yellow to red-yellow to yellow-brown ; instar II., yellow-green to whitish-green ; instar III., light green to green ; instars IV. and V., dark sap-green with whitish wing



1 mm

FIG. 81. *Piesma quadrata* Fieb., adult female.

buds. In all instars the antennæ, proboscis and legs are yellow-brown, the terminal antennal segment darker brown, the terminal segment of labium blackish-brown, eyes bright red to red-brown. All instars show a red or red-brown "abdominal spot" through the abdominal wall (56).

Last Instar. Green; oval; head similar in shape to that of the adult, but with processes of side-lobes of face not meeting in front

of central lobe ; pronotum very transverse, centrally carinated, and transversely rugose, much narrower in front than behind, sides slightly foliaceous, posterior angles rather distinct ; scutellum centrally carinate ; wing-pads and scutellum transversely rugose ; surface of abdomen more or less rugose, segments very distinct ; antennæ similar in shape to those of the adult, but with third joint much stouter and shorter ; tibiæ slightly carinated ; tarsi two-jointed, very similar to those of the adult. Length, $2\frac{1}{4}$ to $2\frac{1}{2}$ mm. (3A).

Adult. Pale dull greyish ochreous, more or less spotted with small darker spots, or uniformly ochreous. Oval, head rugose, with a spine in front of each eye, and the cheeks produced, the processes bent, and converging at the apex ; antennæ with the basal joint very thick, the second slightly narrower and shorter, the third very long and thin, the fourth about equal to the first in length and the second in width ; pronotum very strongly punctured, sides dilated and foliaceous in front, showing several rows of fine meshes, slightly rounded ; base nearly straight ; disc raised posteriorly, with a deep transverse channel in front, which is traversed by three fine carinæ (these are an important diagnostic character), scutellum small, raised, pale, and tuberculate at the apex ; elytra with the corium and clavus punctured like the pronotum ; membrane hyaline, with four parallel nervures, legs ochreous. Length, $2\frac{1}{2}$ to 3 mm. (41).

Food Plants. The food plants belong mostly to the Chenopodiaceæ, and include, besides the sugar beet, *Schoberia salsa*, *Chenopodium* sp., *Salsola kali*, and *Atriplex hastata*. The bug has also been recorded from *Aster tripolium*. It has been found to feed under experimental conditions upon *Polygonum*, *Raphanus*, *Sinapis*, *Thlaspi*, *Amaranthus*, cabbage and spinach. It does not attack potato.

Life History

In Germany the adult bugs hibernate from October till about mid-April. The hibernating quarters usually consist of uncultivated land containing tufts of rough grass and litter, but the bugs may also find shelter in fields of winter wheat and winter rye. By the end of May the hibernated individuals have commenced egg laying, and this is continued till about the end of July, when these bugs die. The eggs are usually laid on the under surface of the young beet leaves and the nymphs emerge in a few days. The developmental periods of the four larval stages are very variable, the total larval stage averaging about thirty-five days. The larva moults five days after hatching and twice again at five-day intervals. The complete life cycle from egg to adult is from four to six weeks in the field. All stages of the insect occur together in the field throughout the summer. The

bugs are full grown by the end of August, and it is these insects which hibernate. There is only one generation a year as a rule, though a second generation has been observed in Germany. In October the insects leave the beet fields and seek out their winter quarters, which are usually close at hand. Few bugs are therefore present in the fields when the beets are harvested. In England the insect matures in August and the adults may be found in that month, September and October. It occurs again in May; the larvae occur in June, July and August.

Ecology of *P. quadrata*. No eggs are laid at temperatures of 37.4° to 42.8° F., and increasing temperature causes an increase in the number of eggs laid. The maximum numbers have been observed at temperatures of 98.6° to 104° F. (37° to 40° C.), and hot, dry weather seems favourable to the development of the bug. High humidity decreases the number of eggs. Cold weather in March and April prolongs hibernation and wet weather in these months retards oviposition, so that the beet plants are already of vigorous growth when the infestation begins. Heavy soils promote infestation and light soils are unfavourable to it.

Virus Transmitted by *Piesma quadrata*

Beta Virus 3, causing leaf crinkle of sugar beet.

Geographical Distribution. *P. quadrata* occurs over nearly the whole of Europe; on the south side of the Mediterranean it is recorded from Tunis, and it extends into Siberia and Turkestan. In Britain it has been recorded from all the coast counties from Norfolk to Cornwall, together with Durham and Cheshire, from the counties of Carnarvon, Glamorgan and Pembroke in Wales, and from both Scotland and Ireland (3A). It is widespread in Germany, and is particularly abundant in Silesia and Anhalt (42).

HEMIPTERA-HOMOPTERA Jassidæ

Cicadula divisa (*sexnotata* Fall.) Uhl. The six-spotted leafhopper.

DESCRIPTION

Head yellow, basal markings roundish; interocular line widely interrupted in the middle; on the forehead a pair of large roundish, subquadrate or oblong black spots; frons with black side lines. Pronotum yellow, sometimes suffused with blackish. Scutellum yellow, sometimes with a subtriangular black spot on each side of the

base. Elytra yellowish-grey, generally subpellucid without markings, sometimes with a greater or lesser number of the areas each occupied by a fuscous streak; membrane sometimes smoky. Abdomen black, yellow at the apex beneath. Legs yellow, with black lines and points. Length, 2.75 to 4 mm.

The markings on the head are very variable in extent, being more prone to vary by way of reduction than in the allied species, and all, or any of them, may be absent (17) (see Fig. 87, A and B).

Food Plants. This leafhopper has a wide range of food plants in the United States, and is a pest of forage, cereals, garden crops and flowering plants. It has been found abundant in meadow and marsh grasses and pastures, and is common on timothy grass in Maine. It is also a serious pest of oats. Kunkel (28) found that *C. divisa* will live and reproduce on aster, lettuce, sow thistle, great ragweed (*Ambrosia trifida* L.), daisy fleabane (*Erigeron annuus* (L.) Pers.), English plantain (*Plantago lanceolata* L.), dandelion (*Taraxacum officinale* Weber), wheat, oats, rye, barley, calendula, *Ammobium alatum*, *Matricaria alba*, *Centaurea imperialis*, *Gaillardia grandiflora*, *Chrysanthemum leucanthemum*, and *Dimorphotheca aurantiaca*.

Life History and Habits

Field observations made in the San Joaquin and Santa Clara valleys in California during winter and spring indicate that the six-spotted leafhopper flies into the cultivated areas after the pasture vegetation becomes dry on the plains and foothills. Spring brood adults are common during March in fields of barley and oats adjacent to the foothills in the San Joaquin valley, but during the middle of April, after the grain begins to ripen, the adults fly to other food plants.

In New York the leafhopper passes the winter in the egg stage, but in California it overwinters as an adult, deposits its eggs, and is at the end of its natural life in March. Nymphs in the last instar may be taken on the foothills of the Santa Clara Valley at the end of February (45).

Viruses Transmitted by *Cicadula divisa* Uhl.

Callistephus Virus 1, causing aster yellows disease.

Callistephus Virus 1A, causing celery yellows disease.

Geographical Distribution. The six-spotted leafhopper is widely distributed in Europe, and also in North America from Alaska to

Florida and from Maine to California. It has also been recorded from Japan.

Jassidæ (continued)

Cicadulina mbila Naudé. The maize leafhopper (see Fig. 82).

TECHNICAL DESCRIPTION

Balclutha mbila Naudé, S. African Journ. Nat. Hist., IV., No. 5, p. 307, 1924. *B. mbila* Naudé, Union S. Africa, Dept. Agric., Ent. Memoirs, No. 4, p. 90, pl. VIII., Fig. 10A-E, 1926.

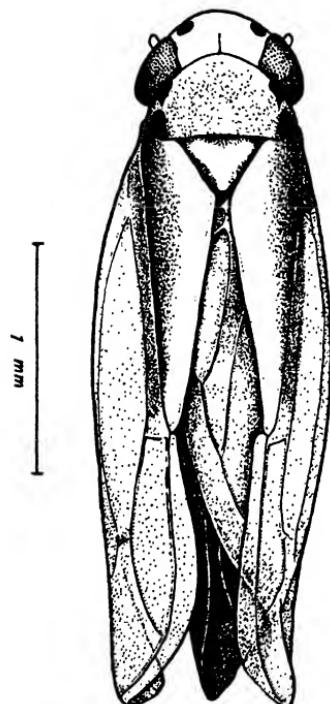
Cicadulina mbila China, Bull. Ent. Research, XIX., pt. 1, p. 61, Fig. 51, and Fig. 2A, 1928.

Colour. Head, pronotum and scutellum pale greenish-yellow, the tegmina greenish-white, sub-hyaline along costal margin cinereous over membrane. Eyes black, two round black spots on anterior margin of head, and a broad dark brown stripe extending from pronotum below each eye down middle of each tegmen, becoming indistinct in region of membrane. Claval commissure narrowly white. Wings hyaline with brown veins. Genæ, pleura, sterna and legs yellowish-white. Dorsum, except for yellow lateral margins, black with white annulations, yellow towards apex; venter greyish-yellow with posterior margins of segments narrowly white. Apex of ovipositor black.

Structure. Head, including eyes, parabolic in outline, its anterior margin obtusely rounded, broader than pronotum, twice as broad as long, and more than half as long as pronotum in middle; ocelli distinct,

placed on the anterior margin of the head close to each eye; antennæ robust, moderately long. Pronotum one and three-fourths as broad as long, rounded anteriorly, broadly emarginate posteriorly, lateral margins feebly convergent anteriorly, almost parallel, posterior lateral corners truncately rounded. Scutellum with straight sides, three-fourths as long as pronotum with a faint transverse impression in middle. Tegmina extending well beyond the apex of abdomen by one-fourth their length, appendix well developed, with only three apical cells, the outer branch of the first sector distinct and forming the outer side of a more or less distinctly closed cell by its union apically with the outer fork of the inner branch of the first sector. Wings with the

FIG. 82. *Cicadulina mbila* Naudé.
The maize leafhopper.



first two veins confluent in their apical third and running into the submarginal vein as one nervure. Face very slightly broader than long; frons strongly convex, twice as long as clypeus; clypeus twice as long as broad, slightly exceeding genæ, apex truncate, base convex, sides slightly concave near base; genæ rounded below, extending by a narrow margin beyond loræ to clypeus.

Male Genitalia. Last ventral segment slightly angulately concave behind, equalling the valve in length. Valve angulately rounded behind; subgenital plates one and a half times as long as valve, subtriangular, contiguous, together parabolic in posterior outline, lateral margins with short white setæ. Parameres relatively broad apically, the pointed apex curved outwards at right angles to the median line of abdomen; lateral spines of pygophor apically straight and acuminate with a long sharp spur projecting at right angles from as far below the apex as its own length; ædeagus narrow tubular with a pair of short lateral anteriorly directed processes in middle and without basal processes.

Female Genitalia. Last ventral segment as long as the penultimate, concave behind with a small central tooth; centre of posterior margin lined with reddish-brown; posterior angles narrow and pointed; ovipositor robust, rounded, almost three times as long as the combined median length of the last two segments. Valves strong, equalling ovipositor, with many strong, white setæ towards apices, which are narrow and pointed.

Length, 3·5 mm.; width, 0·8 mm. (China, W.E., 1937).

POPULAR DESCRIPTION

The eggs are white and about $\frac{1}{60}$ inch long. They are inserted into the leaf substance, and unless laid in the mid-rib are visible from either side of the leaf. The nymphs are white with two black eyes when hatched. They are wingless, but otherwise resemble the adult in shape. Sooner or later a purple mark appears on the upper side of the thorax and abdomen. The portion on the thorax has the shape of a trapezium, and that on the abdomen the shape of a triangle with its apex towards the thorax. This mark may appear after the first moult or after any subsequent moult. The wing pads are to be seen clearly after the third moult. The adult measures about $\frac{1}{60}$ inch in length and is of the characteristic leafhopper shape, widest across the head, and from there narrowing posteriorly. When at rest it appears to be longitudinally striped, having a light yellow line down the back, then a dark band on each side over the thorax and wings, and a narrow light outer edge to the upper wings. The upper side of the body under the wings is black or purplish-black. The colour of the head, legs, antennæ, and under side of the body is light brown or yellowish with a greenish tinge under the abdomen, probably from the contained food. The eyes are black, and in front of the eyes, closer together, are two round black spots (34).

Food Plants. In Natal this leafhopper is commonly found on maize (corn) and sugar cane. It has been successfully fed on Napier fodder grass and *Digitaria* sp., and it is likely that it will thrive on most of the grasses.

Life History and Habits

The following account of the life history is that given by Merwe (34) for conditions in South Africa. The eggs take from nine to twenty-one days to hatch, the longest periods being towards the end of August, while the minimum incubation period occurs between Christmas and the New Year. Eggs laid during January take ten to eleven days to hatch, and apparently after that the

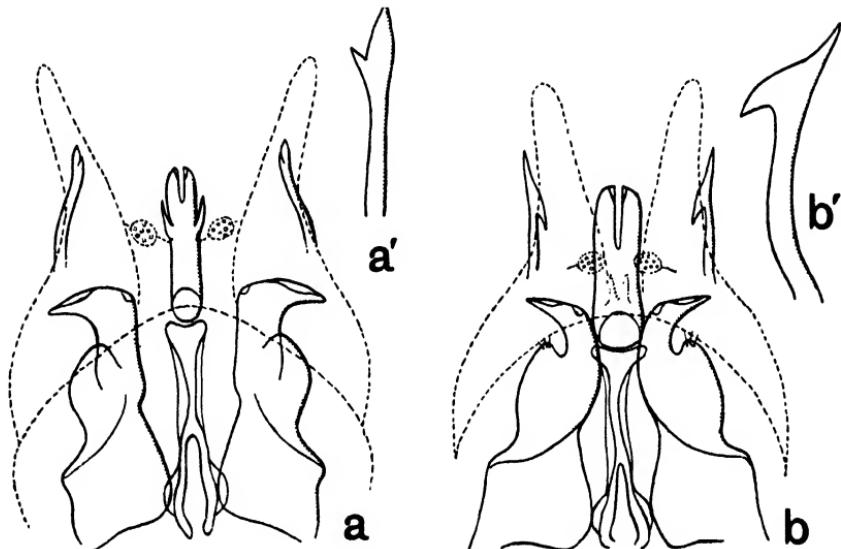


FIG. 83.
Cicadulina storeyi sp. n.
 a. Male genitalia in ventral view.
 a'. Lateral view of spine of pygophor.
Cicadulina zew China, Tanganyika.
 b. Male genitalia in ventral view.
 b'. Lateral view of spine of pygophor.
 (After China.)

egg stage continues to lengthen. During the cold months the stage is probably longer than that recorded for August. The nymph moults five times, reaching the adult stage at the fifth moult, the average length of the nymphal period being twenty-three days. The length of time the adult lives has not been determined accurately, but it probably lives for several months. The female commences egg laying from six to fifteen days after reaching the adult stage. One female was observed to lay 889 eggs in thirty-six days, the greatest number of eggs deposited in one day being twenty-two.

Ecology. The distribution of *Cicadulina* spp. appears to be markedly controlled by climatic factors. Some information upon this point is available from the Amani district, East Africa. The insects are most prevalent on the coastal plains; as the foothills of the Usambaras are ascended they diminish in frequency; while at Amani (3,000 feet) they are extremely rare. *C. milia* disappears first in this progression, whereas the other two species (*C. zæ* and *C. nicholsi*) (see Fig. 83) appear to tolerate better the higher altitudes. It is probable that the distribution of maize streak disease (*Zea Virus* 2) may, in part at least, be related to the distribution of the insect vectors. Streak is severe on the plains, rather infrequent in the foothills and absent from Amani (50).

Viruses Transmitted by *Cicadulina mbila* Naudé

Zea Virus 2, causing streak disease of maize (corn).

Zea Virus 3, causing mottling disease of maize (corn).

Geographical Distribution. *C. mbila* is common in many parts of South and East Africa, and it is probably present in Mauritius since the streak disease of maize occurs there.

Jassidæ (continued)

Euscelis striatulus Fallen. The blunt-nosed leafhopper.

This insect is considered by some authorities to be a separate species, *Euscelis vaccinii* Van Duzee, and it is listed by Ball (1) under the name of *Ophiola striatula* Fallen.

DESCRIPTION

The Egg. The egg is oval-shaped, pearly white, and measures about 1 by 0.3 mm.

The Nymphs. The nymph moults five times during growth. In the first instar it is pale greenish-yellow and is almost transparent during the first day, but then becomes opaque. This instar lasts about four days, and during this period the insect measures from 1 to 1.3 mm. in length. In the second instar the colour is of various shades of green, this period lasts about six days and the insect measures 1.5 to 1.8 mm. in length. The colour becomes a darker green in the third instar, which lasts about seven days. The length of the nymph at this period is from 2 to 2.4 mm. Little change in colour occurs in the fourth and fifth instars except that the insect becomes progressively darker. The length of the insect in the fourth instar is from 3 to 3.4 mm., and in the fifth from 3.4 to 4 mm.

The Adult. Sexes distinct, males dark brown, females more tawny, legs dark, femora twice annulate with pale yellow. Length of female 4.5 mm., male 4 mm., width 1 mm. (see Fig. 84 A, B).

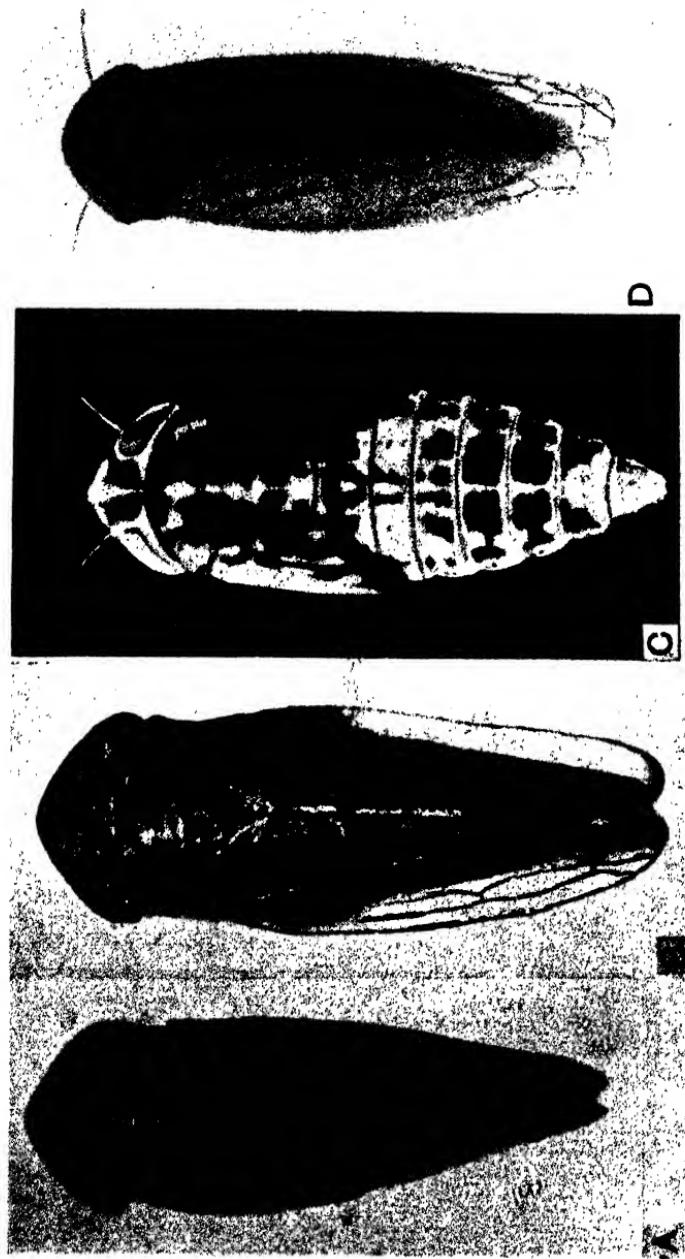


FIG. 84.

Eutettix tenellus.

A. Adult male.
B. Adult female $\times 25$ (approx.).
(After Dobrošeky.)

Euscelis striatulus.

C. Sixth nymphal instar.
D. Adult, summer brood. $\times 30$ (approx.).
(After Severin.)

Colour. Vertex pale yellow with three transverse bands, the posterior one broken forward on each side until it touches the middle one, its medium limb forming a crescent, the median line broadly fuscous connecting the crescent with the band in front; in dark specimens these bands become confluent and the yellow is reduced to elongate spots between them. Pronotum thickly and irregularly marked with fuscous, omitting an elongate spot on the anterior margin. Scutellum dark, usually the margins, a spot on apex and a pair of elongate tri-lobed ones on disc, pale yellow. Elytra light, the inner apical cells smoky, nervures milky-white, the cross nervures very broadly so, nervures broadly, heavily margined with fuscous; in dark specimens often filling up all but a small milk-white spot in the centre of each cell. Face light, with the sutures, arcs on front and a spot on apex of clypeus black; or black with small spots in the middle of the facial pieces and the narrow arcs light. Below dark, anterior and middle femora with two pale yellow bands.

Genitalia. Female segment longer than preceding, bluntly angled and produced laterally and with medium third distinctly produced, lateral margins and pygofer pale yellow. Male valves rounded, almost semi-circular, plates triangular, about twice as long as the valve, a spot on each side of the disc and stout hairs on the margin yellow.

Food Plants. *E. striatulus* occurs mostly on bogland where the favourite food plant is the cranberry, *Vaccinium macrocarpon* Ait. It also occurs on related species of *Vaccinium*, but appears unable to feed on *V. corymbosum* L., the blueberry. It is sometimes found on the huckleberry and in grassland.

Life History and Habits

The winter is passed in the egg stage in the cranberry plant, the egg being inserted longitudinally in the stem beneath the thin outer bark. Usually both ends of the egg are inserted beneath the bark, leaving the centre slightly exposed, but some are inserted at one end only, the remainder of the egg protruding at an angle of about 45 degrees. The eggs hatch in May, and there are five instars in the nymphal life. The first adults appear in the third week of June and are all males. The females do not mature until one or two weeks later than the males. The sexes can be distinguished even as nymphs by the fact that females are light yellow in colour and the males are olive green. The adults may be readily separated as to sex because the female is always larger and of a lighter brown colour than the male. There appears to be only one generation in the year. The insect is most abundant during the hottest months, July and August, and it is most active during the sunniest and warmest part of the day. When disturbed, the leafhopper flies close to the cranberry vines, covering a distance

of 2 or 3 feet at the most, and then settles down on the stem of another plant. It alights usually with head upwards and legs clasping the stem; it rarely alights on a leaf. In feeding it is usually found on the stem near the base of a leaf.

Virus Transmitted by *Euscelis striatulus* Fall.

Vaccinium Virus 1, causing cranberry false-blossom disease.

Geographical Distribution. *E. striatulus* occurs in the United States of America, east of the Rocky Mountains. It is the most common species in cranberry bogs in New Jersey, Massachusetts and Wisconsin. It is not found in Washington and Oregon.

Jassidæ (*continued*)

Eutettix tenellus Baker.

DESCRIPTION

The following description of the insect is taken from the work of Stahl (48).

The Egg. When first laid the egg is transparent, elongate and slightly curved. The posterior end tapers gradually almost to a point. Length, 0.0612 to 0.0696 mm.; average width, 0.0182 mm. As the embryo develops, faint spots which later become conspicuous eye spots appear on either side of the anterior end. During development the colour of the egg changes from white to lemon-yellow with a slight tinge of green.

The Nymph. The recently hatched nymph is nearly transparent, with a light yellow tinge in the thorax and abdomen. The antennæ are hairlike and more than half as long as the body. The head is wider than the thorax or abdomen and is the most distinctive characteristic of this instar. After the first moult the nymph is more slender and the head and antennæ are not nearly so conspicuous. Average length, 1.40 mm.; width, 0.45 mm. Colour usually milky-white with a green tinge. Faint brown blotches may be distinguished on the thorax. In the third instar there is more variation in the colouring; general colour varying from yellow with light brown markings to almost black. The pattern made by the brown blotches does not seem to be constant, but the denser coloration on the thorax has been designated as a "saddle." The colour variations in the fourth instar are similar to those of the third. A red coloration is often observed. The spines on the legs are more conspicuous than formerly, and the wing pads extend to the dorsal margin of the third abdominal segment. Length, 2 to 3 mm. After the fourth moult the nymph has a slender appearance and is nearly the size of the adult. The wing pads extend approximately to the dorsal margins of the fourth abdominal segment. Length, 3.2 mm. (see Fig. 84 C).

The Adult. In California, during the summer, adults of this species show a gradation in colour from light green with no markings to dark

grey with numerous markings on the elytra. In the autumn the percentage of dark forms rises and in the winter the light form is scarce or absent. Some of the winter forms appear almost black.

Light Form. Front yellow, with faint, light brown, transverse stripes. Eyes grey, with occasional brown spots. Vertex green and lemon-yellow, the yellow predominating. Pronotum green; scutum deep yellow; elytra hyaline with light brown venation. No pigment in the elytra. Tergum appearing as dark bands through the folded elytra (see Fig. 84 D).

Dark Form. Front yellow, with irregular, testaceous, transverse bands. Eyes a mixture of red and brown, with red usually predominating. Vertex fulvous, apical portion with a white band cut in the centre by a narrow dark band. Pronotum olive, except for ivory anterior band with several black spots. Scutellum with two square, black spots at basal angles. Elytra sub-hyaline marked with black as follows: two large, almost circular spots on corium; apical portion with irregular black blotches on claval region. Nervures dark brown, with dark pigment on each side forming irregular bands.

Differentiation of *E. tenellus* from *Cicadula divisa* (= *sexnotata* Fall.)

C. divisa may often be confused with the green forms of *E. tenellus*. The six spots on the vertex of the former are usually plainly evident and serve to distinguish it from *E. tenellus*.

Host Plants of *E. tenellus*

Breeding Plants. In the foothills of the San Joaquin Valley in California the leafhopper has been bred from five different species of annual plants. These are *Erodium cicutarium*, red-stem filaree, and *E. moschatum*, white-stem or musk filaree (*Geraniaceæ*); *Hollisteria lanata* (*Polygonaceæ*); *Malvastrum exile* (*Malvaceæ*); and *Lepidium nitidum*, the common peppergrass (*Cruciferæ*). The red-stem filaree is the most important host plant upon which the spring generation develops.

Nymphs also have been observed hatching from eggs deposited in the leaves or stems of the following perennial members of the *Chenopodiaceæ* growing on the plains of the San Joaquin Valley: (*Atriplex fructiculosa*) ballscale, (*A. spinifera*) spinescale, (*A. semibaccata*) Australian saltbush.

Food Plants. During the spring in California the leafhopper occurs in enormous numbers on various short-lived annual saltbushes such as arrowscale (*Atriplex phyllostegia*) and other species of *Atriplex*. In the summer, when these plants become dry, the insects assemble on sugar beet and on other species, notably fogweed or silver scale (*Atriplex argentea expansa*), redscale

(*A. roseæ*) and Russian thistle (*Salsola kali tenuifolia*). A complete list of the food and breeding plants of *E. tenellus* is given by Severin (44).

Life History

The eggs of *E. tenellus* are deposited on a large number of different wild and cultivated plants, most of which belong to the Chenopodiaceæ. The sugar beet, however, is the preferred host plant in the summer season. A single female may lay as many as 247 eggs. The incubation period of the egg varies from ten to fifteen days and the nymphal period from twenty-five to thirty-two days.

In southern Idaho only one generation in the year has been observed, while from two to four may occur in California. In southern Idaho the leafhoppers appear in the beet fields in June and start reproducing at once, oviposition continuing throughout the season. After harvest the insects enter a true hibernation period.

In California the adults appear in the beet fields soon after the beginning of April and remain until harvest time, when they disperse to wild vegetation. No true hibernation has been observed in California (48).

Ecology of *E. tenellus*. In California the spring dispersal of the leafhopper from the uncultivated plains and foothills to the cultivated areas occurs after the pasture vegetation becomes dry and is probably associated with a food stimulus. The dispersal of the summer generation from badly diseased beets to healthy beets is known to extend at least three miles in the Sacramento Valley. These migrations are probably associated with overwintering and unfavourable food. The autumn dispersal from the cultivated areas to the uncultivated plains and foothills in the San Joaquin and Salinas valleys occurs during October, November and December. During the autumn dissemination, the insects congregate on favourable weeds growing on abandoned farms on or near the plains. Frequently the lines of flight across the plains to the canyons and mountain passes follow dry creek beds where the insects take short flights from perennial to perennial. The autumn flights of the leafhopper are also probably associated with a food stimulus; the insects fly from the cultivated districts when the saltbushes and other favourable weeds become woody and dry (44).

The following facts relating to the ecology of *E. tenellus* are

taken from studies made by Carter (4), who investigated the relationship of a number of physical factors to the movements of the insect. A relationship seems to exist between intensity of light and phototropism, and light appears to have some effect on the preoviposition period. Humidities approaching saturation are fatal if maintained for a long time, but such humidities do not ordinarily occur under natural conditions. The insect is unaffected by wind unless disturbed. Carter considers that the introduction of certain host plants into abandoned lands has helped the multiplication of the leafhopper. Further, the Russian thistle (*Salsola pestifer*) is an important host plant in late summer and autumn, and it is the presence or absence of this plant which determines in large measure the distribution and size of the populations of *E. tenellus* which enter partial hibernation. The mustard (*Sophia filipes*), *Atriplex rosea*, etc. are important spring hosts, since it is from these that the migrations appear to take place. The optimum environment for *E. tenellus* with respect to physical factors is found in a sparsely growing crop of Russian thistle (*S. pestifer*). The seasonal development of the host plants governs the migration of the insect in southern Idaho and host plant succession throughout the season is essential if a true permanent breeding ground is to be maintained. Severe winters are followed by reduced population of *E. tenellus*, and consequently good yields of sugar beet in southern Idaho.

Virus Transmitted by *Eutettix tenellus*

Beta Virus 1, causing "curly top" of sugar beet.

Geographical Distribution of *E. tenellus*. This insect has been reported from the following states of North America, as well as from Canada and Mexico: Arizona, California, Colorado, Idaho, Kansas, Nebraska, New Mexico, Oregon, Texas, Utah and Washington. It has also been reported from the greater part of the temperate and subtropical zones of the Argentine Republic, though recent attempts to collect the insect in these parts of the Argentine have failed. Henderson (22) explored Lower California, the west coast and the central district of Mexico, Arizona, Utah and southern Idaho for parasites of the beet leafhopper. He states: "Neither the beet leafhopper nor its parasites were found on the high central plateau, which extends from the southern portion of Durango to Mexico City. Although the territory from Nogales, Arizona, to Mazatlan, Sinaloa, was covered, the range of

the leafhopper apparently extended only as far south as Guasave, Sinaloa."

Jassidæ (continued)

Macropsis trimaculata Fitch (= *Pediopsis trimaculatus* Fitch).

TECHNICAL DESCRIPTION

Nymphs. The nymphs are robust in form, with short transverse head, convex pronotum, and broad abdomen in keeping with other species of the genus. They belong to that group in which the abdomen is lifted into a sharp crest, each segment of which ends in a well-defined tooth which projects backwards. In such characters they differ markedly from the nymphs of *tristis* Van D. Their coloration is a reddish-brown, contrastingly marked with fulvous and quite freely fuscopunctate. Pronotum medially fulvous. Basal angles of the scutellum blackish, a broad fulvous vitta (longitudinal coloured line) contiguous to the scutellar region, and a second just caudad and paralleling it. Wing pads mostly yellowish-green. Seventh, eighth and ninth abdominal tergites fulvous near the crest, the teeth black; and usually a fulvous vitta arises on either side of the abdomen near the origin of the second pair of wing pads and widens posteriorly, ending on the lateral margins of the seventh and eighth tergites. Face dark brown, clypeus and beneath yellowish, thoracic pleurites blackish, legs yellowish, striped with blackish, genital pieces infuscated.

Adults. Fusco-testaceous, the males nearly black, three pellucid white spots in a row on each elytron, clavus cinereous. Length: female, 5 mm.; male, 4.5 mm. Width: female, 1.75 mm.; male, 1.5 mm. (see Fig. 85).

Pronotum parabolically obtuse-angled before, anterior impressions shorter than the long diameter of the eye; rugæ strongly oblique, distinct though fine, median line wanting; posterior margin deeply incised. Face scarcely tumid when viewed from the side, plainly and evenly punctate, rugæ distinct above. Clypeus narrow, longer than the basal width, the sides straight and parallel to the truncate and depressed apex. Loræ broad, strongly tumid. Scutellum finely punctate on the disc. Elytra short, roundly truncate, coriaceous in texture, veins weak.

Genitalia. ♂Edagus of the male flaringly bifurcate at the apex. Last ventral segment of the female triangular, notched at the apex.

Colour. Fusco-testaceous, the males nearly black. Face somewhat lighter, fusco-punctate, superior tip and apex darker, that of the male

FIG. 85. *Macropsis trimaculata* (the plum and peach leafhopper).

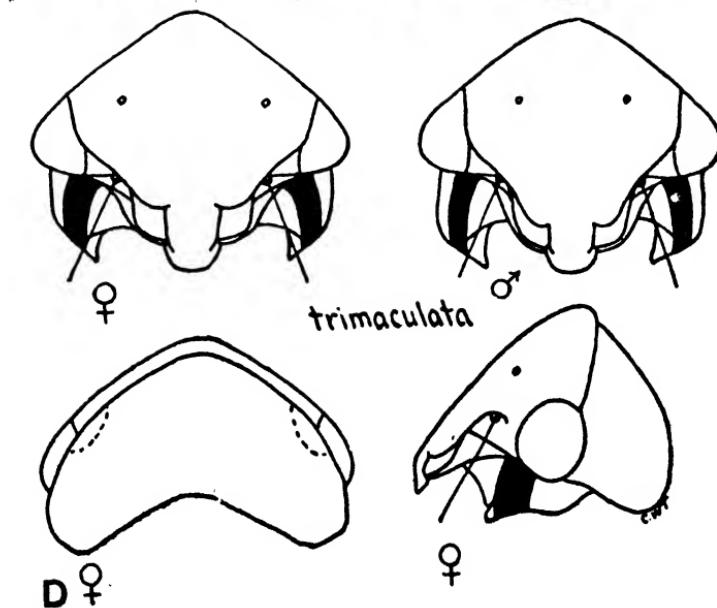
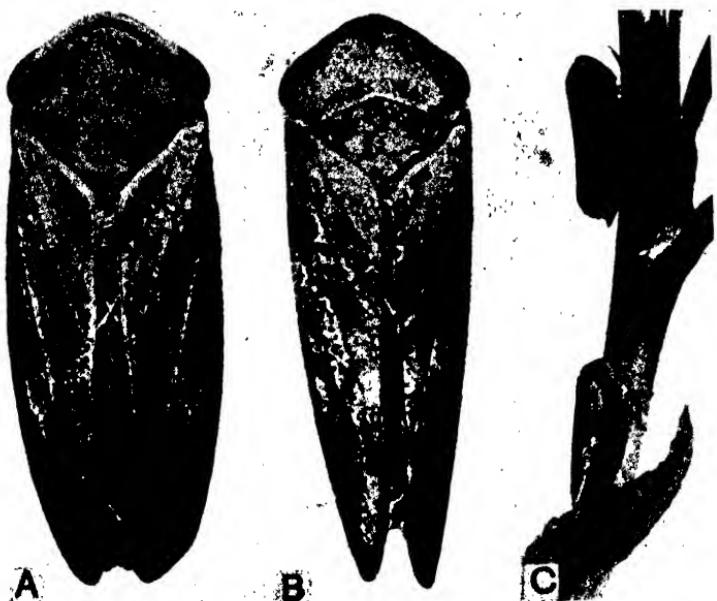
A. Adult female. $\times 15$.

B. Adult male. $\times 15$.

C. Adults feeding on peach stem.

D. Frontal, dorsal and lateral aspects of head.

(A and B, after Hartzell; C, after Kunkel; D, after Breakey.)



surrounded with a whitish halo. Loræ paler, yellowish. Beneath infuscated, the male nearly black, the margins of the segments lighter. Epimera each bearing a black bar which is nearly obscured in the male by the impigmentation. Episternal pieces heavily stained with fuscous. Pronotum of the female testaceous, a dusky patch on the disc ; that of the male fuscous, overcast with cinereous. Scutellum cinereous, fusco-punctate, a dark triangle within either basal angle. Elytra with a pellucid white spot within the third and fourth apical cells, a larger one at the juncture of the ante-apical and basal cells, and a third near the branching of the first sector ; veins pale, clavus cinereous. Wings smoky hyaline, veins fuscous (3).

POPULAR DESCRIPTION

The adults are of a dull reddish-brown colour. The males are usually considerably darker and somewhat smaller than the females. The markings are rather indistinct, but well-marked specimens have three transparent white spots in a row on each elytron. The average length of the females is 5 mm., compared with 4.5 mm. for the males.

The nymphs are robust and are reddish-brown in colour, with short transverse heads and broad abdomens which are lifted in a sharp crest. Each segment ends in a well-defined tooth which projects backward (21).

Food Plants. *M. trimaculata* breeds mostly on plums, the Japanese varieties (*Prunus salicina*) being more infested than the European or the American varieties (*P. domestica* or *P. americana*). It also feeds on peach and apricot, but apparently not on cherry.

Life History and Habits

In New York the nymphal period extends from the latter part of May to the end of the third week in June. The first nymphs appear about May 28th, and become adult about the middle of June, the nymphal period thus lasting approximately three weeks. Adults occur in the field from the middle of June till the middle of August. The life of an individual adult insect is about twenty-seven days. The winter is passed in the egg stage in the bark of the wild plum and there is only one generation in the year.

M. trimaculata rarely feeds on the foliage, but seeks the twigs and smaller branches (see Fig. 85, C). It has been observed feeding for the most part on young twigs and only occasionally on the petioles and midribs of the leaves. The nymphs and adults run rapidly and seek the opposite side of the limb from the observer. This, combined with their colour, which resembles the bark of the tree, makes them difficult to collect. They rarely take to flight and seldom hop, though they may do so on occasion (21).

Viruses Transmitted by *Macropsis trimaculata*

Prunus Virus 1, causing the yellows disease of peach, and, probably, **Prunus Virus 1A**, causing the little-peach disease.

Geographical Distribution. Widely spread over the United States of America, it has been collected in Colorado, Iowa,

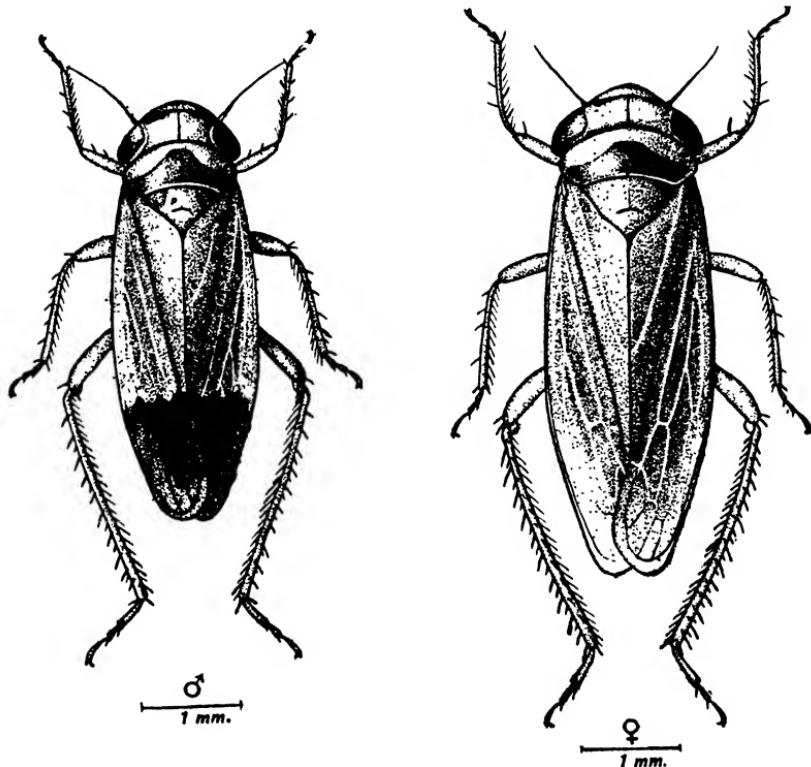


FIG. 86. *Nephrotettix apicalis cincticeps* Uhler (= *N. bipunctatus cincticeps* Uhler). (After Fukushi.)

Illinois, Minnesota, Wisconsin, Michigan, Ohio, Pennsylvania, New York, Ontario and Maine. It probably occurs in all the states from Nebraska eastwards where the peach is grown.

Jassidae (continued)

Nephrotettix apicalis Motsch., var. *cincticeps* Uhl. The rice leafhopper.

DESCRIPTION

Egg. Yellowish-white in colour, elongate, slightly curved and tapering at one end. Length, 1 mm.

Nymphs. The larvæ are at first creamy-white and hairy with some-what indistinct black stripes along both sides of the body. As they grow various minute black spots and an irregular pattern of dark colour develop on the back of the thorax and abdomen. The dark coloured nymphs grow into males and the light coloured ones into females.

Adult. The adult is yellowish-green in colour with a length of 4·5 to 6 mm. and an average width of 1·6 mm. across the broadest part of the head, the male being smaller than the female. Dorsally the vertex is yellowish-green with a black transverse mark on the anterior margin; pronotum and scutellum green; elytra yellowish-green, with black distal ends in the male and with the latter slightly tinged with grey in the female; abdomen black. Ventrally, the thorax and abdomen are light brown in the female and black in the male (19) (see Fig. 86).

Food Plants. Chiefly the rice plant, but also *Astragalus sinicus* L., and various wild grasses.

Life History

In Japan this leafhopper is a four- or five-brooded insect, and oviposition extends over a considerable period, making the seasonal history rather complicated. The eggs are deposited in the tissues of the leaf-sheaths, usually fifteen to twenty-five in one spot, and are thrust transversely into the tissues lying one under the other. As a rule the leafhopper overwinters as a nymph, feeding on *Astragalus sinicus* and wild grasses; it becomes mature in April or May. Later the adults migrate to the rice seed beds and begin at once to feed on the seedlings. If the season is sufficiently advanced, egg laying begins at about the same time and continues over a long period, each female depositing more than 100 eggs. The eggs hatch in seven to thirteen days and the larvæ mature in about twenty days. The leafhoppers continue to breed on the rice fields after the rice seedlings are transplanted and three or four generations of the insect are produced there, the first adults of each brood developing in June to July, August and September respectively. The eggs of the final brood of the season hatch in September and October, and the nymphs hibernate among *Astragalus sinicus* or in grasses growing in the rice fields (19).

Virus Transmitted by *N. apicalis*, var. *cincticeps*

Oryza Virus 1, causing dwarf disease of rice.

Geographical Distribution. This leafhopper, like the virus it transmits, appears to be confined to Japan.

Jassidæ (continued)

Peregrinus maidis Ashm. (*Pundaluoya simplicia* Dist.). The corn leafhopper.

DESCRIPTION

Body and legs ochraceous, lateral margins of the abdomen alternately spotted with yellow and black : femora piceous or piceous-brown, their apices and the tibiae and tarsi pale ochraceous ; face with the lateral margins and a central line bifurcating anteriorly, pale castaneous ; lateral margins of clypeus pale castaneous ; antennæ ochraceous with the apex of the basal joint piceous ; eyes piceous ; vertex with the lateral margins and two central fascia testaceous ; pronotum with four central testaceous fascia which terminate before the anterior and posterior margins ; mesonotum with four testaceous fascia ; abdomen above, in some specimens, considerably shaded with piceous ; tegmina hyaline with a yellowish tint, the veins yellow, with apical black markings, principally on apical margin, surrounding marginal cellular areas, and on the oblique veins at apex of costal area ; wings hyaline with faint opaline lustre, the veins pale fuscous (14).

Length, excluding tegmina, 2.5 mm. ; expanded tegmina, 10 mm.

Food Plants. The main host of this species is maize or corn, *Zea mays*, but it also occurs on native grasses.

Life History and Habits

This insect is closely related to the sugar cane leafhopper (*Perkinsiella saccharicida*), but in Hawaii it is found only on maize. In confinement it will oviposit in stems of sugar cane and *Coix lachryma*, but the nymphs apparently cannot develop on these plants. When it is abundant on maize, the plant withers as if suffering from drought. The hoppers are distributed mainly by flight and also by hopping from adjacent fields and by wind dispersion. The eggs are deposited principally in the mid-rib on the upper surface of the leaves, from one to four being loosely packed in each cavity. These hatch in summer in nine days, but in cold dry weather may take fourteen days. The nymphal stage lasts from fifteen to twenty-one days, during which five molts occur. Dimorphism is frequently found among the adults, both long- and short-winged forms occurring. Mating and oviposition take place as soon as the adult insects appear, each female depositing an average of 200 eggs. The life cycle occupies about one

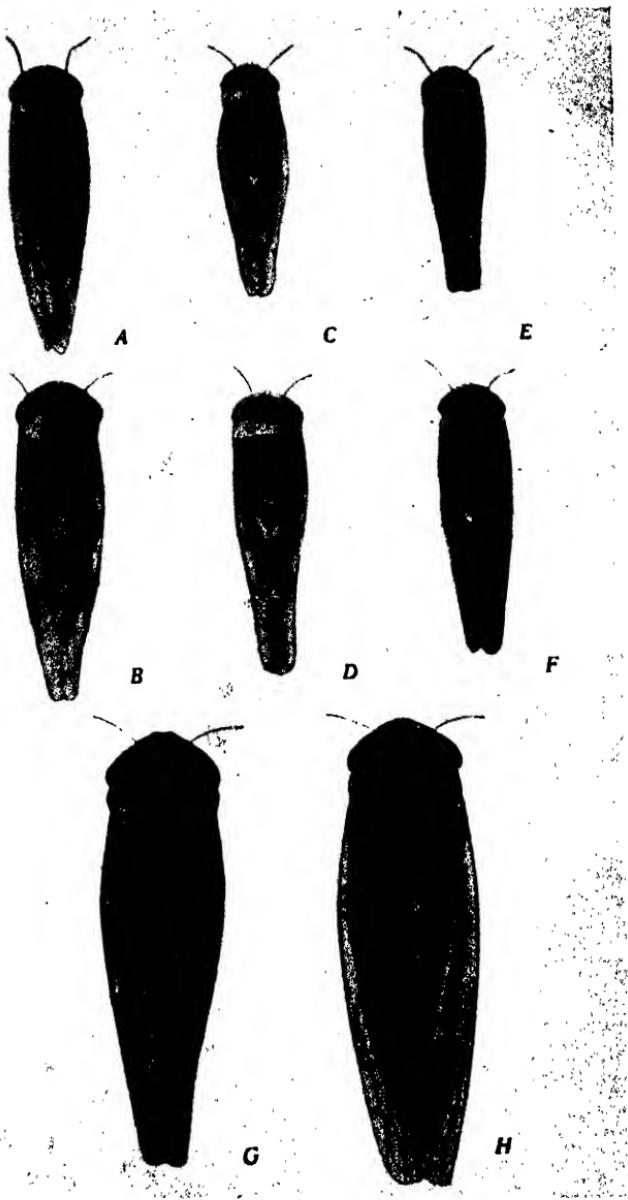


FIG. 87.

A, B. *Cicadula divisa*.

C, D. *Thamnotettix montanus*, adults of summer generation.

E, F. *Thamnotettix montanus*, adults of autumn generation.

G, H. *Thamnotettix geminatus*. (After Severin, *Hilgardia*.)

month, and as maize plants require from 100 to 120 days to mature, it is obvious that several generations of hoppers may be present on the plants simultaneously (20).

Virus Transmitted by *Peregrinus maidis* Ashm.

Zea Virus 1, causing stripe or mosaic disease of maize (corn).

Geographical Distribution. North America, Hawaii, Fiji, Australia, Java, Amboina, the Philippines, Formosa, Malay Peninsula, British India, Ceylon, Seychelles, West Africa, Cuba, Nicaragua and Brazil.

Jassidæ (continued)

***Thamnotettix montanus* Van D.** The mountain leafhopper.

This insect is 4·5 to 6 mm. long, with white or yellowish face, a transverse brownish band between the eyes, a conspicuous yellow transverse band on the pronotum, and the scutellum brown. Severin reports that summer adults collected in the northern San Joaquin, southern Sacramento, Santa Clara and Salinas valleys in California were dark brown, while specimens taken during the autumn were usually black, with intermediate stages between the two colour patterns. The insect is common on grasses, weeds, carrot, larkspur, golden rod, apple and prune. It has a wide range of food plants in California and has been collected abundantly on celery (45) (see Fig. 87, C-F).

Virus Transmitted by *Thamnotettix montanus*

Callistephus Virus 1A, causing celery yellows disease.

Geographical Distribution. *T. montanus* has been recorded from British Columbia, Washington, Oregon, California, Nevada, Idaho and Colorado.

Jassidæ (continued)

***Thamnotettix geminatus* Van D.** The geminate leafhopper.

The insect measures 5 to 6 mm. in length, and is greenish-yellow or brown in colour, with a pair of black spots on the anterior border of the head, a black spot on each side of the eye, an arched band near the front border of the pronotum and black spots on the scutellum. It occurs, sometimes in large numbers, on clover, lucerne (alfalfa), grasses, grains, apple, ~~carrot~~ and aster (see Fig. 87, G and H).

Virus Transmitted by Thamnotettix geminatus
Callistephus Virus 1A, causing celery yellows disease.

Geographical Distribution. *T. geminatus* has been recorded from Colorado, Idaho, California, Washington and Alaska (45).

HEMIPTERA-HOMOPTERA
Delphacidae

Delphacodes striatella Fall. (*Liburnia striatella* Fall.).

DESCRIPTION

Male. Black ; facial keels, pronotum (except a large spot behind each eye) and apex of scutellum whitish, crown pale. Elytra but little longer than abdomen, pale brownish, rounded at the apex, veins sparingly granulate. Legs yellow. Aperture of pygofer constricted, its outline sinuate, upper notch semi-circular, its margin, seen from the side, oblique ; hind margin nearly vertical, sinuate just before it joins the margin of the upper notch. Styles very short, broad at the base, rapidly narrowing towards their somewhat rhomboidal apex. Teeth of anal tube long, vertically subcontiguous. Length, 1·75 to 2·75 mm.

Female. Pronotum and scutellum widely pale down the middle, their sides and the abdomen above blackish, the latter sometimes with pale spots towards the apex. Length, 2·5 to 3 mm.

Macropterus (long-winged) form. Elytra hyaline, nearly twice as long as abdomen ; veins fine, pale brown, darker towards the apex, sparingly granulate, apical third of costal vein black, sixth apical area fuscous, at least in the female. Length, 3·5 to 4·5 mm. (15) (see Fig. 88, 1-3).

Food Plants. Various wild grasses in the British Isles and the rice plant in Japan.

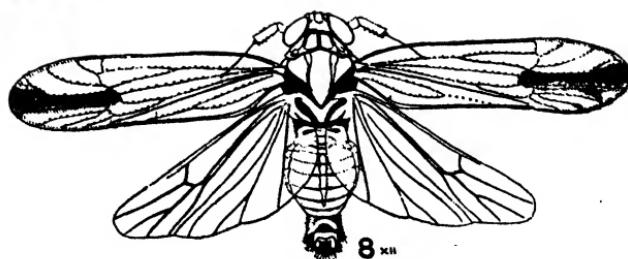
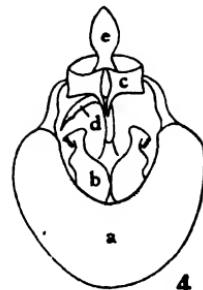
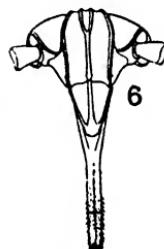
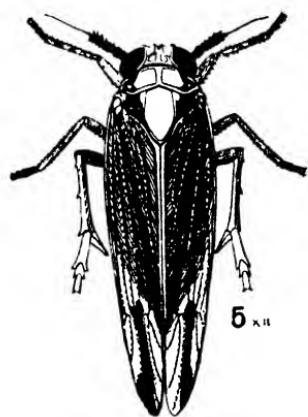
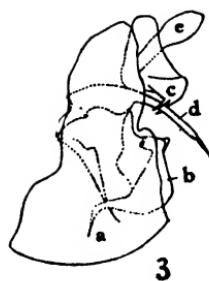
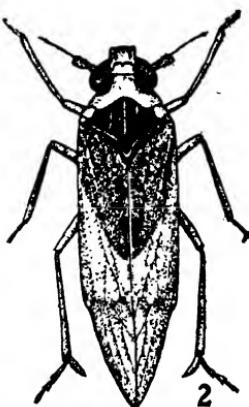
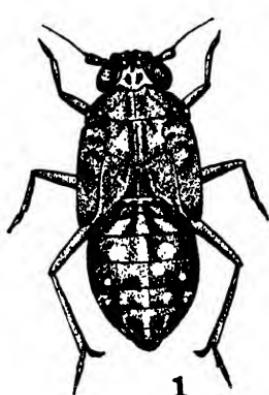
Virus Transmitted by Delphacodes striatella
Oryza Virus 1, causing dwarf disease of rice.

FIG. 88.

Delphacodes striatella Fall.

1. Larva in fifth instar (darker form).
2. Adult male.
3. Male genital segments in profile.
4. Male genital segments, postero-ventral view.
5. Adult female, macropterus form.
6. Face, adult male.
7. Genital segments, adult male.
8. Adult male.

(1-4, after Teiso Esaki and Shiro Hashimoto, 5-8 ; after Kirkaldy.)



Geographical Distribution. The British Isles and probably Europe generally ; it also occurs in Japan.

Delphacidæ (continued)

Perkinsiella saccharicida Kirk. The sugar cane leafhopper.

TECHNICAL DESCRIPTION

Adult (Male). Pallid yellowish testaceous ; abdomen above and beneath black, apical margins and laterally more or less widely pallid ; apical half of first segment and carinate (ridged) edges of second segment of antennæ, flagellum, basal half of frons (except the pustules) and a cloudy transverse band near the apical margin of the same, longitudinal stripes on femora, coxæ spotted or banded near the base, a large spot on each pleuron, anterior and intermediate tibiæ with two or three annulations, apical segment of tarsi, etc., blackish or brownish. First genital segment large, deeply acute-angularly emarginate above (see Fig. 88, 4-8).

Adult (Female). Like the male, but abdomen above and beneath stramineous (straw-yellow), irregularly speckled with brownish ; ovipositor, etc., blackish.

Length, male and female, 4.5 mm.

Long-winged Form, Male and Female. Tegmina elongate, narrow, extending far beyond apex of abdomen, interior half of clavus and corium more or less faintly smoky, a long dark smoky stripe on middle of membrane, three or four nervures of the latter smoky at apex.

Length to apex of elytra, 6.5 mm.

Short-winged Form, Male. Tegmina reaching only to base of fifth segment, costa more arched, apex more rounded, neuration similar, but shortened. Tegmina hyaline, colourless ; nervures pale testaceous brownish, with blackish-brown non-piligerous dots (in both forms) (26).

POPULAR DESCRIPTION

The general colour of the adult is greyish-brown. The two basal joints of the antennæ, which stand out conspicuously, are dark brown, as are also the sides of the prothorax ; the abdomen is variegated with spots of darker brown. The wings are semi-transparent with darker areas towards the tips of the forewings. Two forms of the adult female occur—a long-winged summer form and a short-winged winter form—the latter being more prolific than the former. On the frontal margin of the two forewings appears a whitish line which, when the wings are neatly folded together, gives the appearance of a yellowish straw-coloured stripe running along the back of the insect (85).

Food Plants. In its host plants, the insect seems mainly confined to the sugar cane, though it may also feed on wild grasses.

Life History and Habits

The eggs of *P. saccharicida* are deposited beneath the epidermis of the cane plant in situations along the mid-rib of the leaves, in the internodes of the stalk, or, in the case of young unstripped cane, in the leaf-sheath of the lower leaves. When deposited in the leaves, the eggs are inserted from either side, but usually from the inside, the greater number being in the larger portion of the mid-rib down toward the leaf-sheath. The place of incision is indicated at first by a whitish spot, this being a waxy covering over the opening. Later, the egg punctures become more readily discernible owing to invasion by fungi which cause a reddish discolouration. The female is capable of laying about 300 eggs in the course of her life, from four to six eggs being deposited in each puncture. The nymphs hatch during a period of two to five weeks after oviposition, according to season, and pass through four nymphal stages before attaining the adult stage in an additional period of about thirty-four days, the life of the adult being usually about one month. Preference is shown by the insects for succulent vigorously growing canes, and they usually select the younger immature leaves in the spindle, in the folds of which they also tend to escape detection by their many natural enemies. In Queensland the leafhoppers increase rapidly in numbers throughout January, February and March, a peak period being reached during March to May, after which the population declines. Stunted and poorly growing canes are not favoured by the insect for oviposition. Although able to complete its life cycle on wild grasses, the leafhopper exhibits a decided preference for sugar cane, and in Queensland is rarely found on other plants in the vicinity of canefields.

Ecological Studies. Secondary spread of the virus disease of which this leafhopper is the vector has been observed in Queensland (35) to be always greater in areas where the cane is growing vigorously, either on account of greater rainfall or irrigation, or increased soil fertility. On the evidence of population counts, the relation between total leafhopper population and the vigour of the cane in each field is remarkably consistent. In other words, the numbers of leafhoppers are greatest in the hotter and wetter months of the year and in areas where growing conditions are best.

Virus Transmitted by *Perkinsiella saccharicida*
***Saccharum Virus 2*, causing Fiji disease of sugar cane.**

Geographical Distribution. *P. saccharicida* is indigenous to Australia, where it occurs throughout the sugar cane areas. From there it was introduced some time prior to 1900 into Hawaii. It has also been recorded from Java.

HEMIPTERA-HOMOPTERA Psyllidæ

Paratrioza cockerelli Sule. The potato and tomato psyllid.

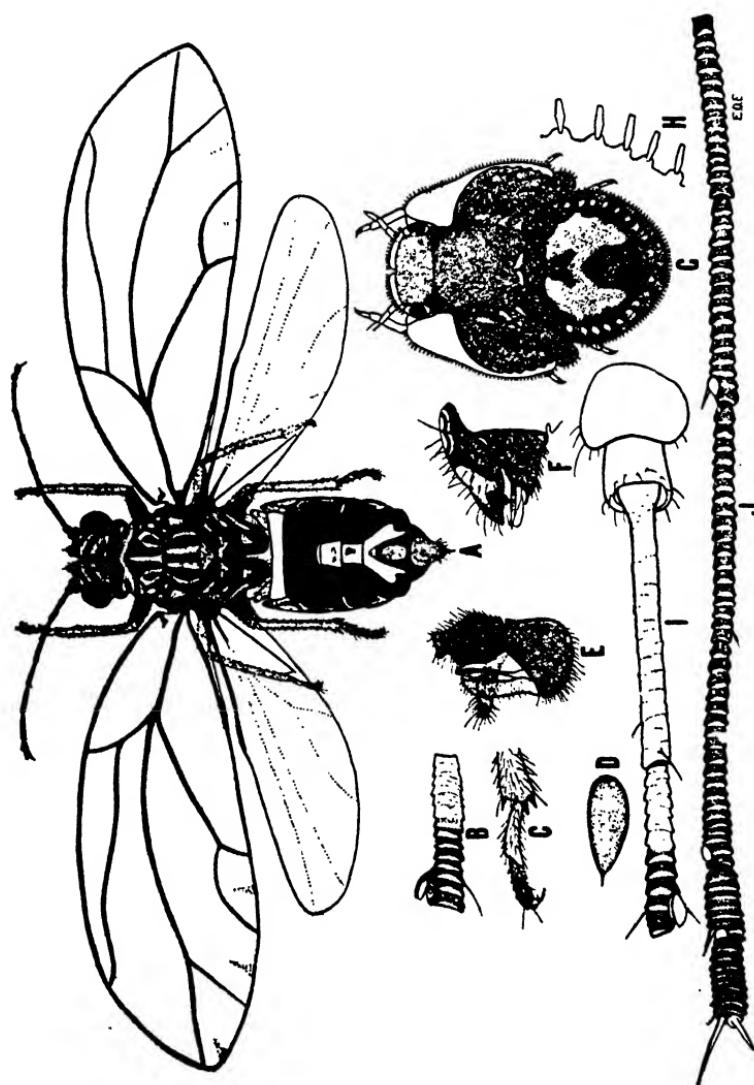
TECHNICAL DESCRIPTION

Head. Breadth of the vertex behind the eyes, 0·34 mm., with the eyes 0·52 mm., length along the middle line 0·16 mm., posterior margin of the vertex regularly and slightly excised, posterior angles slightly truncate, the anterior ones broadly rounded ; a median line divides the vertex in two halves, either showing a distinct fovea (shallow depression) in the middle. Antennæ filiform, 1 mm. in length ; sensoria present on joints II., IV., VI., VIII., IX., that on the fourth joint being in the form of a hollow ball with a circular opening, covered with a spoon-shaped movable operculum ; the other sensoria are in the form of simple pits. The three basal joints of the antennæ are yellow, IV. to VIII. yellow, brown at the ends, the last two black. Frontal lobes very short, 0·06 mm. long, rounded at the apex. Clypeus in the form of a half-pear, not produced anteriorly. Head colour, ground, yellow-white, in the middle of the vertex a large, brown, horse-shoe shaped spot ; middle line of the vertex and frontal lobes dark brown, the apex of the latter being lighter, clypeus yellowish-white. Thorax, ground colour, yellowish-white, with a large well-defined brown mark. Elytra, length, 2·60 mm. ; greatest breadth, 1 mm. ; nervures fine, yellowish-white, transverse nervules colourless. Membrane vitreous ; no spines on either side of the elytra membrane. Hindwings and legs as usual in this genus.

Apex of Male Abdomen. Genital segment equal in breadth and height, 0·17 mm. ; posterior and inferior margin evenly arcuate, anterior half of the upper side moderately arcuate ; no tubercles, hairs

FIG. 89. *Paratrioza cockerelli* Sule. The tomato psyllid.

- A. Adult female with wings spread.
- B. Fourth joint of the antenna showing sensorium with covering or operculum.
- C. Tip of tibia showing apical spines.
- D. Egg.
- E. Genitalia of the male.
- F. Genitalia of the female.
- G. Nymph.
- H. Fringe of spines round margin of nymph's body (greatly enlarged).
- I. First four joints of the antenna.
- J. Last six joints of the antenna. Much enlarged.
(After Essig.)



of moderate size, equally scattered on the surface, colour black; forceps in the form of a scythe, with its edge anteriorly; posterior margin straight, anterior sinuate; height, 0·17 mm.; breadth, 0·04 mm.; hairs uniformly on the surface, colour brownish. Anal segment 0·17 mm. high, produced posteriorly in a triangular lobe; very long setæ, especially on its distal end; colour brown.

Apex of Female Abdomen. Anal segment viewed from above, short, triangular, obliquely truncate at the end; viewed laterally, upper margin slightly arcuate, rounded at the end; inferior margin straight, slightly excised before the apex; long hairs scattered on the sides; on the upper margin and just under it, short, strong, acute spiniform hairs, before the apex a few long stout hairs, and around the anus, short small hairs; on the whole surface of the segment are very small short, acute spines in rows parallel with the posterior margin. External sheath narrowing behind with roundly truncate apex, reaching over the end of the anal segment. The inner stylets straight, obliquely truncate on the under side before the apex; apex itself acute with two triangular teeth above. The innermost stylets triangular at their end. Colour of anal and genital segments dark brown, sometimes with a few whitish spots.

Length, 2 to 5·3 mm. to the end of closed wings (51) (see Fig. 89).

POPULAR DESCRIPTION

Egg. The egg is exceedingly small, elongate-oval, with the attached and pointed end supported by a short petiole and the free end broadly rounded. The colour is transparent white or pale greenish-yellow with a more or less definite orange coloured mass at the middle or base. The surface is normally entirely covered with a fine white powdery wax which gives a decidedly grey appearance. The powdery wax is also deposited over the surface of the leaves around the eggs and aids their discovery. They are usually deposited in large numbers upon the under surfaces of older leaves and stand erect or slightly leaning. Length, 0·08 mm.

Nymphs. The first instar nymphs are very small and of a transparent pale yellow with orange coloured head and abdomen. The colour changes somewhat as the insects mature, and when ready for the last moult they are pale greenish-yellow with grey and orange markings on the dorsum. Except for the fringe of scale-like spines round the margin, the bodies are smooth. The shape is flat and broadly oval and the body is held close to the surface of the leaves while feeding.

Adults. The mature insects are at first pale green or light amber in colour, but soon become darker, there being a considerable variation in colour. Normally the colour is light amber brown with numerous very dark brown or black markings. A very conspicuous white powdery stripe extends across the dorsal base of the abdomen, and a definite horseshoe-shaped and quite large area of the same colour, and covered with fine white powder, occurs on the dorsal posterior two-thirds of the abdomen. The legs are light amber and sparsely hairy. At the tip of the hind tibæ there are two apical dark spines on the inside and one on the outside. The antennæ are ten-jointed and dusky, excepting the first three and the basal half of the fourth joints. In many

individuals the bases of all the joints, excepting the last two, are yellow. On the fourth joint near the tip is situated a circular sensorium with a peculiar lid or operculum, while on each of joints VI., VIII. and IX. there is a single, simple, circular sensorium near the tip.

Length of body about 1·8 mm. (18).

Food Plants. The host plants are imperfectly known, but the insect has been recorded on the following: Lucerne (alfalfa), tomatoes, and many solanaceous plants such as potatoes, petunia, *Nicotiana* spp. and *Datura* spp.

Life History and Habits

The winter is passed in the adult stage upon evergreen food plants or in sheltered places. The eggs are laid late in April and until late autumn in the mild climate of California. The broods are uneven and all stages of the insect may be found from May until November. There appear to be at least three, and probably more, overlapping broods in the year.

Virus Transmitted by *Paratrioza cockerelli*

Solanum Virus 18, causing psyllid yellows disease in potatoes.

Geographical Distribution. The insect has a wide distribution in California, and has been recorded from Colorado, Utah, Arizona and New Mexico.

HEMIPTERA-HOMOPTERA

Aleyrodidae

Bemisia gossypiperda M. and L. The cotton white-fly.

DESCRIPTION

Egg. Sub-elliptical, apical end narrower, pedunculate, the stalk is inserted into the tissue of the leaf, chorion smooth, colour light yellow when fresh, subsequently changing to dark brown, the distal free end is of deeper colour. Length, 0·168 to 0·176 mm.; breadth, 0·08 to 0·09 mm.; average measurements, 0·171 × 0·082 mm.; stalk, 0·016 to 0·024 mm. long; average, 0·019 mm.; reddish eye spots conspicuous before hatching and distinctly divided.

Nymph of First Instar. Elliptical, light yellow. Length, 0·248 to 0·256 mm.; breadth, 0·184 to 0·186 mm.; average measurements when newly hatched, 0·253 × 0·129 mm.; margin surrounded by waxy bands and beset with sixteen pairs of bristles, three cephalic—the last cephalic pair being the longest, ten laterals, sub-equal, three caudals—the anal pair being the longest. Two pairs of entral spines—the anterior pair in front of the rostrum and the posterior pair at the level

of the dorsal vasiform orifice. The dorsal spines which are present in the later stages are absent. Antennæ thin and long, 0.066 to 0.078 mm. in length; average, 0.070 mm.; three segmented—basal segment thick, second cylindrical and the third segment elongated and provided with two spines—one at a distance of two-thirds of the length of the segment from the base and the other at the tip. Eyes distinctly divided. Legs functional, measuring 0.07 mm. and reaching beyond the margin of the body when extended; tibia armed with a long curved bristle, tarsus single-jointed with a bristle near the distal end, longer than the tarsus; pedunculated disc-like pad at the end of the tarsus. Mycetoma orange-yellow. Vasiform orifice slightly wider than long; length, 0.020 to 0.028 mm.; breadth, 0.024 to 0.030 mm., average measurements, 0.024×0.027 mm.; operculum semi-circular; lingula setose, twice as long as the operculum and therefore projecting beyond it.

Nymph of Second Instar. Body elliptical, depressed, pale greenish-yellow in colour. Length, 0.312 to 0.350 mm.; breadth, 0.192 to 0.200 mm.; average measurements, 0.319×0.196 mm. Margin crenulate with a waxy band all round. Eyes divided, antennæ much shorter than in the previous stage, directed backwards, 0.021 mm. long. Legs degenerate, unsegmented conical stumps, each ending in a disc-like sucker. Mycetoma orange-yellow. Vasiform orifice, measuring 0.030×0.032 mm., lingula armed with a pair of long hairs at the tip. Dorsal bristles absent or one to three pairs, marginal bristles, only one pair of anals.

Nymph of Third Instar. Shape corresponding to the previous instars. Length, 0.48 to 0.60 mm.; breadth, 0.32 to 0.36 mm.; average measurements, 0.52×0.34 mm. Eyes not completely divided; antennæ directed towards the median line, hook-shaped, the convexity of the hook, antero-medium, 0.020 mm. long. Legs as in the preceding instar. Vasiform orifice, 0.051×0.48 mm. Dorsal spines and mycetoma as in the second instar.

Pupa. Body slightly convex, 0.6×0.4 mm. to 0.8×0.6 mm., colour deep yellow, eyes well developed, not completely divided. Antennæ better developed than those in the second and third instars, 0.056 mm. long, crenulate, thick and pointing backwards and outwards, each terminates in a slightly bent, thin process. Legs curved, unsegmented. Vasiform orifice longer than broad. Length, 0.066 to 0.083 mm.; breadth, 0.050 to 0.058 mm.; average measurements, 0.077×0.052 mm. Dorsal spines variable, when a full complement of seven pairs of dorsal spines is present, the arrangement is as follows: I. Cephalic, pre-ocular; II. post-ocular; III. lateral, at the level of the middle legs; IV. lateral, just behind the above; V. dorso-medial; VI. postero-lateral; on the sides of third and fourth abdominal segments; VII. vasiformal on the side of the vasiform orifice. Besides these there is a pair which is anal (see Fig. 90, A).

Adult Female. Length, 1.1 to 1.2 mm.; body yellow, with two pairs of concolorous wings; eyes constricted in the middle, forming two parts, lower facets bigger than the upper. Antennæ seven-segmented, elongated; segment I. sub-globose, II. sub-pyriform, III. elongated, longest of all and beset with sensoria distally, IV. cylindrical, V. clavate, VI. cylindrical, VII. elongated and imbricate, provided with sensoria

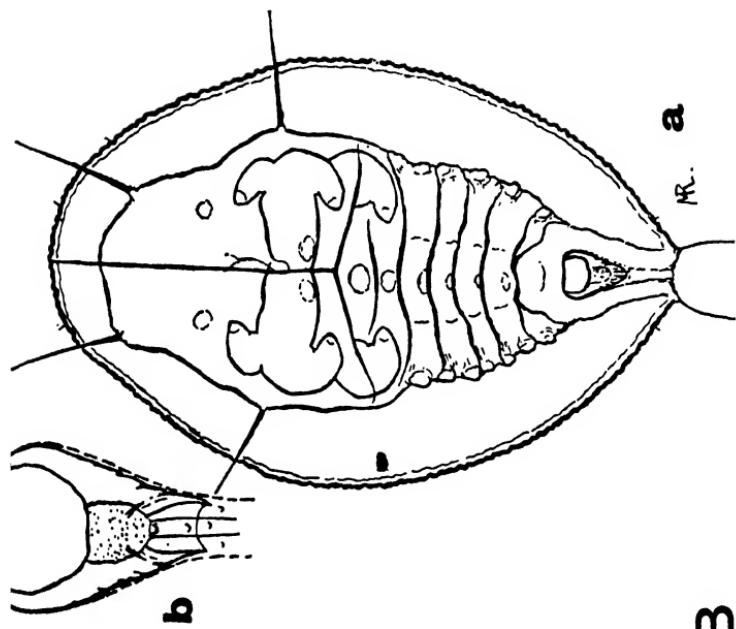
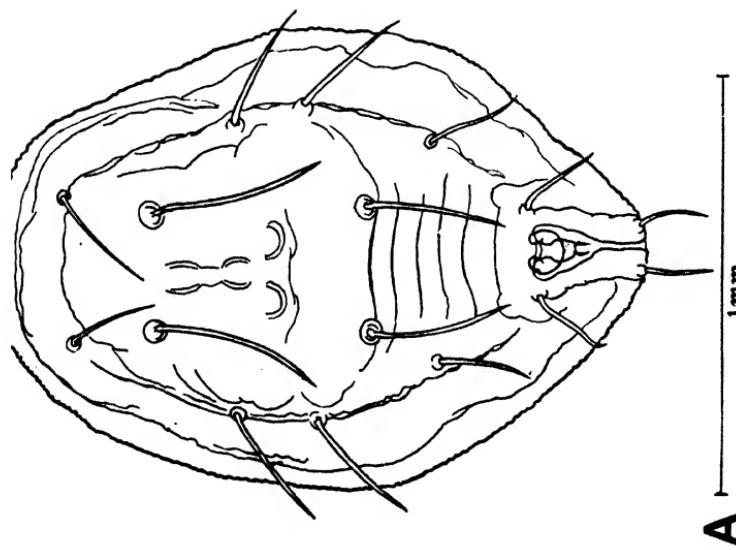


FIG. 90.
A. *Benisia gossypiperda*, Misra and Lamba Pupa case.
B. *Benisia nigriensis* sp. n. a. Pupa case. b. Vasiform orifice.
(B, after Corbett.)



and terminating in a spine. Labium 0·39 mm. long, forewing measuring on an average $0\cdot89 \times 0\cdot34$ mm., venation poorly developed—radial sector and a thin cubitus, hindwing measuring $0\cdot74 \times 0\cdot28$ mm. Hind legs longer than others, tibia 0·32 mm. long; tarsus-proximal joint, 0·112 mm.; distal joint, 0·072 mm.; tibia of front leg, 0·21 mm.; tarsus-proximal joint, 0·080 mm.; distal joint, 0·064 mm.; paranychium acute. Mycetoma orange-yellow. Vasiform orifice with a rectangular operculum and lingula exposed, much elongated and armed with hairs on the tip.

Male. Length from vertex to the tip of claspers, 1·06 mm. Concolorous with female. Antennæ same as for the female, slightly shorter, wings corresponding to those of female, fore wing measuring on an average $0\cdot84 \times 0\cdot28$ mm., hind wings $0\cdot70 \times 0\cdot23$ mm. Abdomen tapering posteriorly; claspers narrowing distally with tips curved inwards and beset with minute bristles. Penis slightly curved, smaller than claspers, measuring on an average 0·080 mm. (23).

Food Plants. Husain (23) records *B. gossypiperda* from the following species of Malvaceæ in the Punjab, *Gossypium* spp., cotton; *Althea rosea*, hollyhock; *Corchorus tridens*; *Hibiscus esculentus*, lady's finger; *Sida cordifolia*.

In addition the insect has been recorded from various members of the following families: Cruciferæ, Cucurbitaceæ, Leguminosæ, Euphorbiaceæ, Solanaceæ, Compositæ, Nyctaginaceæ, Amarantaceæ, Chenopodiaceæ, Convolvulaceæ, Capparideæ.

Life History

The following facts refer to the life history of the insect as it occurs in India. The eggs are laid singly and almost invariably on the under side of the leaves, mostly on the top and middle leaves of the plant. The oviposition period varies from two to eighteen days. The incubation period in the egg varies from three to thirty-three days, temperature being the chief controlling factor. During the main cotton-growing season, i.e., from April to September, the egg stage lasts from three to five days, during October and November it is prolonged from five to seventeen days, and during February and March it is seven to sixteen days. The longest incubation period so far observed was during December to January, when it occupied thirty-three days. The duration of the three nymphal stages varies from nine to fourteen days from April to the end of September, but from October onward this period is considerably prolonged, ranging from seventeen to seventy-three days. In *B. gossypiperda* the pupal period is very short and occupies only two to eight days. A complete life cycle may occupy from fourteen to 107 days. During April to September

it occupies from fourteen to twenty-one days. The shortest life cycles have been observed during August. From October onward the life cycle is much prolonged, and in one case it extended to ninety-seven days between November and February, and the longest life cycle so far recorded, at Lyallpur, has been 107 days. Emergence occurs, as a rule, during the day time. During the summer the adults do not live very long and in captivity two to five days may be considered as their average life. During November, however, some adults may live as long as twenty-four days in cages. Parthenogenesis is common among the Aleyrodidae, and has been observed in this species, only males developing from unfertilised eggs.

Seasonal History. The adults of the first brood of the year commence emerging from about the middle of January. Infestation, as a rule, starts on such weeds as *Convolvulus arvensis* and *Euphorbia* spp., and such cultivated plants as *Brassica rapa* and *B. oleracea*. From these the insect spreads to *Hibiscus esculentus*, the cucurbits and the ratoon cotton. No sooner has the cotton crop germinated in April than the white-fly appears on it and reproduces freely throughout the summer months. The maximum infestation of cotton occurs during July and August, more particularly in the latter month, and then drops suddenly in September and October and after that continues, but is much abated. About the end of the cotton season the insect migrates to *Brassica* spp., and to various weeds and cultivated crops where the immature stages pass the winter. In all, there may be twelve generations of the white-fly in the course of a year, but, as the broods overlap, practically all stages are met with throughout the year.

A study of the seasonal activities of *B. gossypiperda* shows that there are three phases of migration in the course of a year : (1) From cotton to the winter alternative hosts, (2) from there to the spring alternative hosts, and (3) from the latter back again to the cotton. Cucurbits and ratoon cotton are regarded as dangerous sources of infection for the new cotton. The period of quick multiplication of the white-fly coincides with that of germination and growth of the cotton crop. The severity of attack on cotton extends from June to August, while the attack on ratoon cotton continues to be severest till about the middle of July. Infestation on weeds during the off season of cotton suggests that the insect might be checked to some extent by practising clean cultivation and removal of weeds (24). This also

applies to *Nicotiana Virus 10* (tobacco leaf-curl virus), which is transmitted by the same insect ; in this case it has been found in Java that the removal of three species of weeds is important, since they serve to carry the virus over the period that the tobacco crop is not grown.

Viruses Transmitted by *B. gossypiperda*

Nicotiana Virus 10, causing leaf-curl in tobacco.

Gossypium Virus 1, causing leaf-curl in cotton.

Geographical Distribution. *B. gossypiperda* has been recorded from India, where it is widely distributed in the Punjab. It is also present in Pusa (Bihar and Orissa). What is thought to be the same species is recorded from the Sudan, Southern Nigeria, East Africa, Iraq and Java.

Aleyrodidæ (continued)

***Bemisia nigeriensis* sp.n.**

DESCRIPTION

Puparia on leaf without marginal secretion ; dorsal disc and two pairs of spines on cephalothorax prominent. Subdorsal ridge, sides of caudal groove, and abdominal segments yellowish-green, with rest of case paler in colour. Pupa-case elliptical, without a cephalo thoracic constriction ; margin crenulate, without thoracic fold. Dorsal disc distinctly defined by a ridge running round the case. In addition to this ridge, about eight pairs of subcircular, indistinct, blunt tubercles on abdominal segments. Sutures distinct to subdorsal ridge ; first two abdominal segments in median line with a defined subcircular porous area ; the third, fourth, fifth and sixth with an indication of a similar area, and a pair of similar areas mid-dorsally on second thoracic segment. Two pairs of prominent spines, which are apparently easily broken, are placed anteriorly and laterally near subdorsal ridge of cephalothorax. A conspicuous pair arising from tubercles near posterior margin is also present. Vasiform orifice longer than caudal furrow with its anterior margin straight and with about three small tubercles near its outer posterior margin. Inner margin armed with about seven pairs of slender teeth. Lobes surrounding caudal furrow well defined, extending beyond vasiform orifice and meeting in mid-dorsal line. Operculum slightly wider than long, sub-semicircular and not quite filling half of orifice. Lingula with a small projection from tip and not reaching posterior margin of case (5) (see Fig. 90, B).

Bemisia nigeriensis resembles *B. inconspicua* Quaint., from which it may be distinguished by the position of the spines as well as by the marked ridge differentiating the dorsal disc.

Food Plants. *Manihot utilissima* ; *M. palmata*, var. *aipi*, cassava.

Virus Transmitted by *B. nigeriensis*

Manihot Virus 1, causing mosaic of cassava.

Geographical Distribution. This insect appears to have been recorded only from Nigeria.

HEMIPTERA-HOMOPTERA

Aphididæ

Amphorophora rubi Kalt. The raspberry aphid.

DESCRIPTION

Alate Viviparous Female. Green, of various pale shades ; thoracic lobes pale reddish-brown. Slightly hairy. Antennæ much longer than body ; thin, rather dark except base of segment III. and I. and II., which in some, however, are dark, arising from large frontal tubercles ; segment I. much larger than II. ; III. longer than IV., with forty-two to forty-eight sensoria all over it ; IV. a little longer than V., the latter with normal sub-apical sensorium ; VI. with long, thin flagellum about as long as IV. plus V. Rostrum green, apex dusky, reaching to third coxae. Eyes red. Cornicles longer than cauda, thin, but swollen towards middle or apical half, green in some ; green tipped with black in others, a few all dusky ; three striae across apex. Cauda green, rather long and prominent ; with seven pairs of lateral hairs and two median dorsal ones near apex. Legs long and thin, especially hind pair ; green ; apices of tibiae and tarsi dark. Wings large with yellow insertions ; stigma yellowish-grey ; veins almost deep brown.

Length, 3 to 3·5 mm. (see Figs. 91, 2 and 92, 2).

Apterous Viviparous Female. Shiny green of various shades ; slightly hairy. Eyes reddish-brown. Antennæ long and thin, as long as or longer than body ; green ; joints darkened, in some almost black ; there is a dusky patch where the flagellum joins the basal area of segment VI., arising from prominent frontal tubercles ; segment I. is larger than II. ; III. longer than IV., with eight to fourteen sensoria on its basal half ; IV. longer than V. Cornicles long and narrow, slightly expanded at base and also in the middle or towards the apical half ; in some dusky at apex and much longer than the cauda. Cauda green, long, with six pairs of lateral hairs, the apical pair curved at their ends. Legs long, green ; tarsi dusky to black, apices of tibiae dark, but not to the same extent as in the alate female (Theobald, 52).

Length, 3·2 to 3·8 mm.

Food Plants. *Epilobium montanum* (Walk.), *Geum urbanum* (Walk.) ; *Rubus fruticosus*, blackberry ; *R. idaeus*, raspberry ; *Spartium scoparium*, broom.

Life History

A. rubi occurs sometimes in great numbers on raspberries, where it feeds on the under sides of the leaves. In the British Isles this

aphis is most numerous on the raspberry in May and June, but also occurs right through the year. On the bramble it chiefly affects the young shoots and often swarms up them as well as on the lower surface of the leaves. Winged broods are much more frequent on the bramble than on the wild and cultivated raspberries. The ova, at first green, soon become black and shiny ; they are laid singly and very slowly, under the rind of the canes, usually near a bud and some in the axils of the buds. Although the insect lives permanently on the wild raspberry in this country, just as it does on the bramble, the presence of the aphis on the cultivated varieties undoubtedly is due mainly to alate females coming from the blackberry. There is a red form of *A. rubi* which is only a variety. According to Buckton, the broom (*Spartium scoparium*) is an alternate host, but Theobald states that he has never been able to find the aphis on this plant.

Viruses Transmitted by Amphorophora rubi

Rubus Virus 1, causing green mosaic of raspberries.

Rubus Virus 2, causing yellow mosaic of raspberries.

Allium Virus 1, causing yellow dwarf disease of onions.

Geographical Distribution. Fairly common in the British Isles, and probably in Europe generally, it has been recorded from Belgium and Germany. It appears to be widely distributed in the United States of America.

Aphididæ (continued)

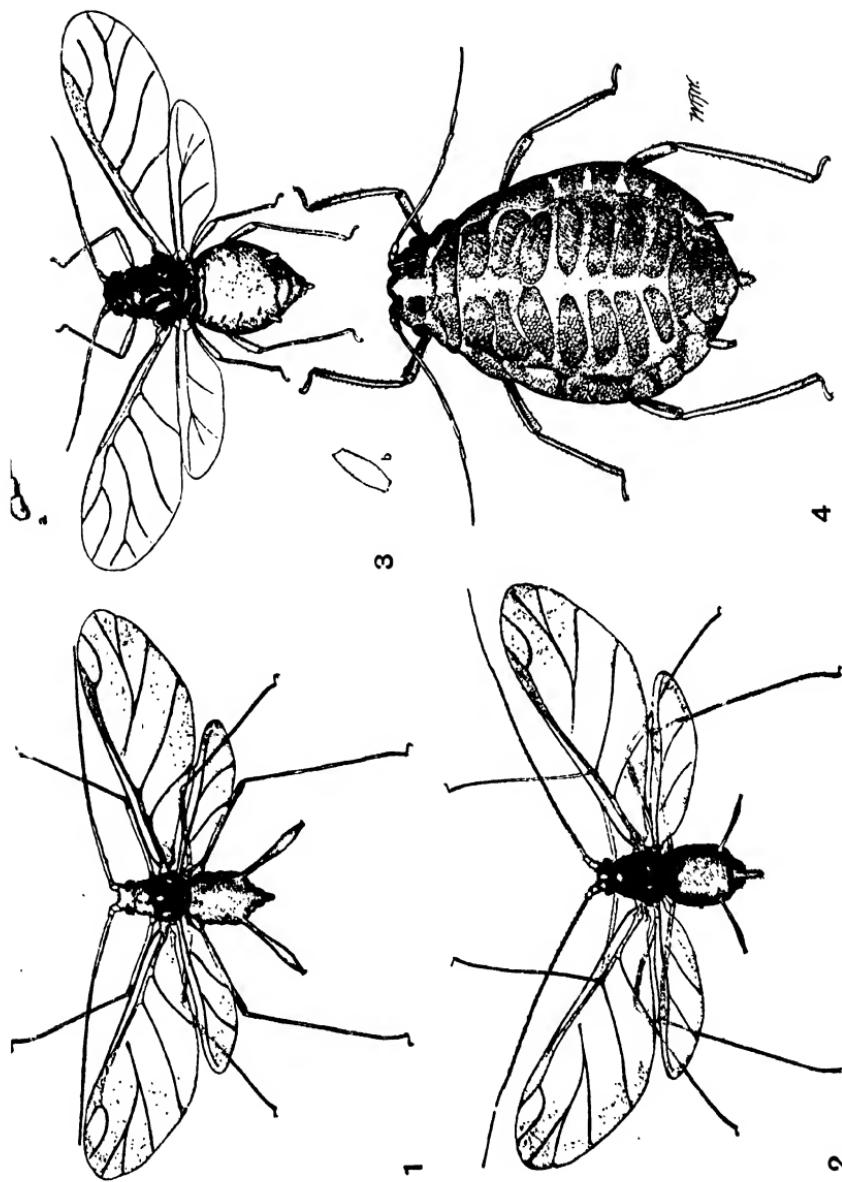
Amphorophora rubicola Oestl.

DESCRIPTION

Alate Viviparous Female. Antennæ about as long as body, dark coloured, except base of III., hairs conspicuous, about as long as width of segment, III. with twenty to thirty sensoria scattered over nearly the entire length and not in a straight row. Antennal tubercles fairly large. Beak extending usually to third coxae. In some specimens there are two tubercles showing on the posterior, dorsal portion of the head.

FIG. 91.

1. *Amphorophora rubicola* Oestl., alate viviparous female.
2. *Amphorophora rubi* Kalt., alate viviparous female.
3. *Rhopalosiphum brassicae*, alate viviparous female :
a, antenna ; b, cornicle of same enlarged.
4. *Rhopalosiphum brassicae*, apterous viviparous female.
(1 and 2, after Mason ; 3 and 4, after Davis.)



The prothorax has two dorsal tubercles, and a lateral tubercle on each side. The front wings have a dusky spot at the tips. This is darker in some specimens than in others. The abdomen has several lateral tubercles, one specimen showing five in front of the cornicle and one caudad of the cornicle. Hairs are present around each tubercle. The cornicles are long, curved, conspicuously swollen and distinctly reticulated. The cauda is long, slender and constricted, with five to six lateral hairs (see Figs. 91, 1, and 92, 1).

Apterous Viviparous Female. Antennæ about as long as body, light coloured except distal ends of segments and VI., hairs numerous, about as long as width of segment, III. with thirteen to fifteen sensoria on basal half. Beak reaching nearly to third coxa. The only tubercles showing are the lateral prothoracic ones. Cornicles very long, not as conspicuously reticulated as in the alates, somewhat imbricated. Cauda small, somewhat constricted (31).

Food Plants. The host plants are restricted to *Rubus* spp.

Life History and Habits

Very little is known of the biology of this species. It is found on the leaves and shoots of various species of *Rubus*.

Viruses Transmitted by *Amphorophora rubicola* Oestl.

Rubus Virus 1, causing green mosaic of raspberries.

Rubus Virus 2, causing yellow mosaic of raspberries.

Geographical Distribution. In North America from Maine to California ; Ottawa and British Columbia.

Rankin (40) gives the following key to the common species of *Amphorophora* feeding on the raspberry, adapted from Mason's monograph (31).

Alate Viviparous Females

(1) Cornicles distinctly reticulated. Distinct dusky spot on tip of wing. Feeding on the canes, *A. rubicola* Oestl.

(2) Cornicles not reticulated, often imbricated at the tip.

(a) Fourth segment of antennæ with sensoria, *A. sensoriata* Mason.

(b) Fourth segment of antennæ without sensoria, *A. rubi* Kalt.

Apterous Viviparous Females

(1) Cornicles distinctly reticulated. Third segment with thirteen to fifteen sensoria. Feeding on the canes, *A. rubicola* Oestl.

(2) Cornicles not reticulated, often imbricated at tip.

- (a) Third segment of antennæ half as long again as the cornicles.
More than twenty sensoria, *A. sensoriata* Mason.
- (b) Third segment of antennæ shorter. Less than twenty sensoria, *A. rubi* Kalt.

Aphididæ (continued)

Amphorophora sensoriata Mason.

DESCRIPTION

Alate Viviparous Female. A large species, general colour green; antennæ longer than body, dark coloured, imbricated, hairs inconspicuous, much smaller than in *rubi*, numerous sensoria on III., IV. and some on V. Antennal tubercles large. Beak very short, in some specimens not reaching second coxae. Prothoracic and abdominal tubercles not showing. Cornicles fairly long, moderately swollen, the tips imbricated, but not reticulated. Cauda of medium length, broad, not constricted, with about three sets of lateral hairs.

Apterous Viviparous Female. Antennæ about a third longer than body, imbricated, the hairs inconspicuous, much shorter than width of segment, III. with a row of sensoria. Antennal tubercles large. Beak reaching about to second coxae. Cornicles moderately long, plainly swollen, the tips imbricated, but not reticulated. The cauda broad, conical, not constricted, with about three sets of lateral hairs (31) (see Fig. 92, 3).

Life History and Habits

Mason records finding this species sparingly on the stems of raspberry, never on the leaves. Pergande says in his notes that aphides of this species are found on the stems of *Rubus*, which they sometimes cover for a distance of several inches. They drop readily if disturbed. The aphis probably remains on *Rubus* throughout the year.

Viruses Transmitted by *Amphorophora sensoriata*

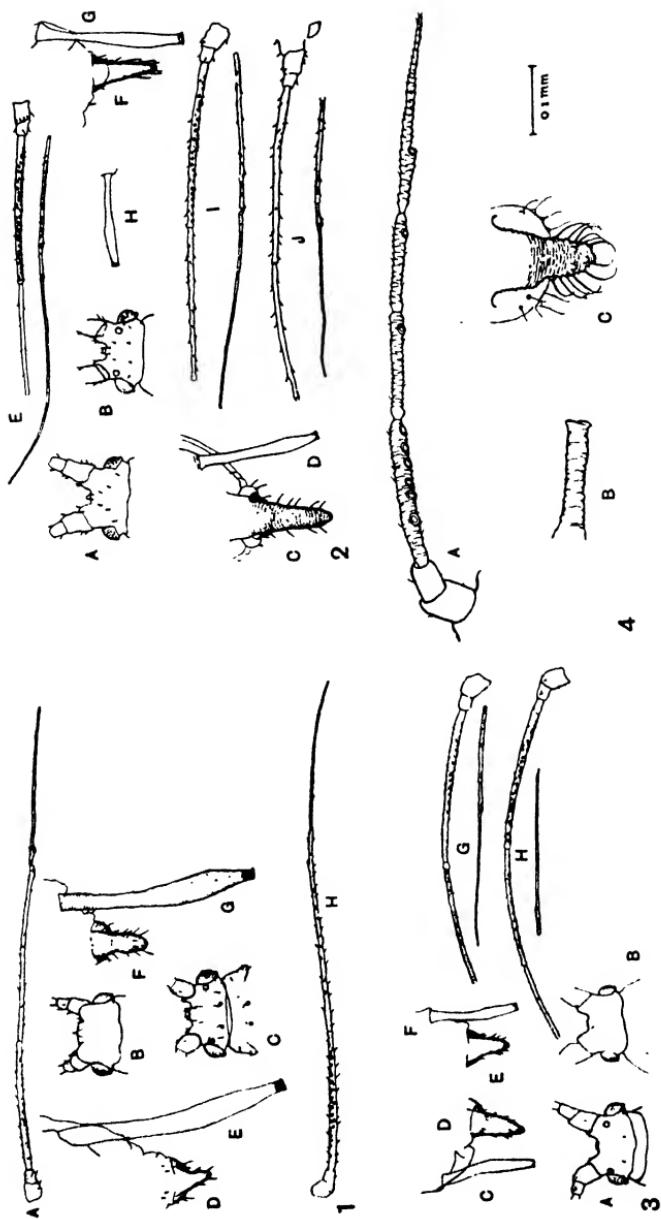
Rubus Virus 1, causing green mosaic of raspberries.

Rubus Virus 2, causing yellow mosaic of raspberries.

Geographical Distribution. Mainly a North American species; it has been recorded from Massachusetts, Pennsylvania, Maryland, District of Columbia, Virginia, West Virginia, Ohio, Minnesota and Kansas.

Aphididæ (continued)

Anuraphis padi Linn. (*Anuraphis helichrysi* Kalt., *Aphis pruni* Buckt.).



There seems to be a good deal of confusion as to what should be the correct name for this aphid, but Theobald (52) considers that it should be known as *Anuraphis padi* Linn. It is a very variable species and descriptions are only given here of the common forms occurring on *Prunus*.

DESCRIPTION

Apterous Viviparous Female (second stage on *Prunus*). Green, more or less shiny, some olive brown to almost ochreous-yellow. Oval, often attenuated caudally. The shiny skin is often coated with a fine mealy substance. Eyes brown. Antennæ, cauda and anal plate green, the former dusky at apicea. Cornicles very small, pale brown to olive green. Rostrum pale green, apex darkened. Occasionally the abdomen has darker lines running lengthwise. Antennæ very short, only about one-quarter length of body, but now and then in later stages one-third ;

FIG. 92.

1. *Amphorophora rubicola* Oestl.
 - A. Antenna, apterous form.
 - B. Head, apterous form.
 - C. Head, alate form.
 - D. Cauda.
 - E. Cornicle, apterous form.
 - F. Cauda.
 - G. Cornicle, alate form.
 - H. Antenna, alate form.

2. *Amphorophora rubi* Kalt.
 - A. Head, apterous form.
 - B. Head, alate form.
 - C. Cauda.
 - D. Cornicle, apterous form.
 - E. Antenna, male.
 - F. Cauda.
 - G. Cornicle, alate form.
 - H. Cornicle, male.
 - I. Antennæ, alate form.
 - J. Antennæ, apterous form.

3. *Amphorophora sensoriata* Mason.
 - A. Head, alate form.
 - B. Head, apterous form.
 - C. Cornicle.
 - D. Cauda, apterous form.
 - E. Cauda.
 - F. Cornicle, alate form.
 - G. Antenna, apterous form.
 - H. Antenna, alate form.

4. *Aphis rubicola* Oestl. (*rubiphila*) Patch.
 - A. Antenna.
 - B. Cornicle.
 - C. Cauda
 - (all alate form).

(1, 2 and 3, after Mason.)

segment I. larger than II. ; III. longer than IV., shorter than VI. ; IV. slightly longer than V. ; base of VI. about as long as V. ; flagellum twice or more length of base. Rostrum reaches second coxae. Cornicles short and thick, expanding basally ; no imbrication. Anal plate and cauda hidden under abdomen as a rule, but in fully mature specimens the latter may project ; two hairs each side of cauda. A few hairs on posterior of body.

Length, 1·6 to 2 mm.

Alate Viviparous Female (on *Prunus*). Head and thorax dark brown to black ; lobes shiny black. Abdomen green, with black dorsal patch posteriorly, one dark transverse line usually in front of it and two behind ; four dark spots each side. Antennæ deep brown to black. Legs yellowish-green, apices of femora and tibiae and the tarsi dark. Cauda green. Cornicles dark green, apices dark, some paler than others. Sterna of thorax black and a black spot at apex of body on venter. Wing insertions pale ; stigma greyish-green ; veins pale brown. Antennæ nearly as long as body, segment I. broader than II. ; III. long, about as long as VI., with twenty-nine to thirty-five sensoria ; IV. much longer than V., with thirteen to eighteen sensoria, some larger than others ; V. not quite so long as base of VI., the latter about one-quarter of the flagellum. Rostrum reaches second coxae. Cauda bluntly elongate, three hairs each side and one dorso-apical, about as long as cornicles. Anal plate rather flattened, rounded apically, many hairs. Cornicles short, cylindrical, not so long as antennal segment V., no imbrications. Tibiae with small hairs. Head and body with very short scattered hairs. (Theobald, 52).

Length, 1·4 to 1·7 mm.

Food Plants. All kinds of plums, damsons, sloes, hawthorns, peaches and other *Prunus* spp., and many kinds of herbaceous plants, *Chrysanthemum* spp., various *Umbelliferae*, *Rumex*, *Cineraria*, *Vinca*, etc.

Life History and Habits

Usually called the leaf-curling plum aphid, this is one of the commonest aphides found on plums, damsons, etc. The sexual forms occur on *Prunus* in autumn, and the oviparous females lay their eggs at the bases of the buds and among the bud clusters. The eggs hatch in late February and early March. The young from these eggs are at first dull green, but later turn into fat purplish-brown aphides which shelter at the bases of the buds in characteristic manner. These produce green young which in their turn give rise to numerous progeny which feed on the leaves and cause them to curl. In late May and early June the green apteræ give rise to alate forms which leave the *Prunus* and feed on various different herbaceous plants, such as *Myosotis* spp., *Umbelliferæ*, *Senecio*, *Trifolium*, etc.

Virus Transmitted by Anuraphis padi

Prunus Virus 5, causing mosaic of *Prunus* spp.

Geographical Distribution. Widely distributed over Great Britain and Ireland, Continental Europe, South America, North America, Japan, India and Egypt.

Aphididæ (continued)

Anuraphis tulipæ Boyer (*Aphis tulipæ* Boyer; *Aphis gladioli* Felt.).

DESCRIPTION

Alate Viviparous Female. Head brown; thorax dark brown to black, lobes varying from very deep brown to black; abdomen pale fawn to almost white, a few yellowish of various shades, or tinged with pink; dark dorsal and lateral markings, notably a varied-shaped dark dorsal patch on segments IV. to VI. or VII. Antennæ, cauda and anal plate fuscous, former with some pale or yellow areas. Legs pallid, now and then with yellowish tinge; apices of femora and tibiæ dusky; tarsi dark, venter pallid yellow or white, a dark area on the mesonotum; apex of abdomen with fuscous bars. Antennæ shorter than body; segment I. larger than II.; III. about as long as VI., rather thick, with forty-eight to sixty sensoria over whole length; IV. wider and a little longer than V., with three to fourteen sensoria; VI., with basal area not quite so long as V.; flagellum shorter than III. Eyes large, deep red. Rostrum reaches well past third coxæ. Cauda small; two hairs each side and one dorso-apical. Anal plate rounded, with many hairs. Cornicles rather short, cylindrical, narrow, varying in shape, some expanded basally, but always with a marked constriction one side of base, about one and a half to twice as long as cauda and about as long as or longer than hind tarsi. A marked blunt papilla behind posterior legs on each side and a small one before cornicles. Two pairs of small sub-median dorsal papillæ at apex of abdomen. Stigma grey; veins pale greenish-brown; insertions pallid.

Length, 1.7 mm.

Apterous Viviparous Female. Pale fawn to almost white, a few tinged with pale pink or brown, yellowish-brown or pallid green; head and apex of body may be deeper coloured. Antennæ pale, apices dark, also dusky bands on apices of segments III. to V. Cauda, anal plate and cornicles fuscous. Apices of tibiæ and tarsi dusky. Eyes black to deep red. Rostrum pale, apex dusky. Antennæ less than half body length; segment I. larger than II.; III. about as long as VI., more than twice as long as IV.; IV. longer than V.; basal area of VI. smaller than V.; flagellum about as long as IV. plus V.; several prominent hairs on III. Cauda small, scarcely projecting beyond abdomen, with two hairs each side and one dorso-apical. Anal plate rounded with several long hairs. Cornicles cylindrical, expanding basally, about three times as long as cauda and nearly as wide at base, much longer than hind tarsals. Rostrum reaches third coxae. Abdominal lateral

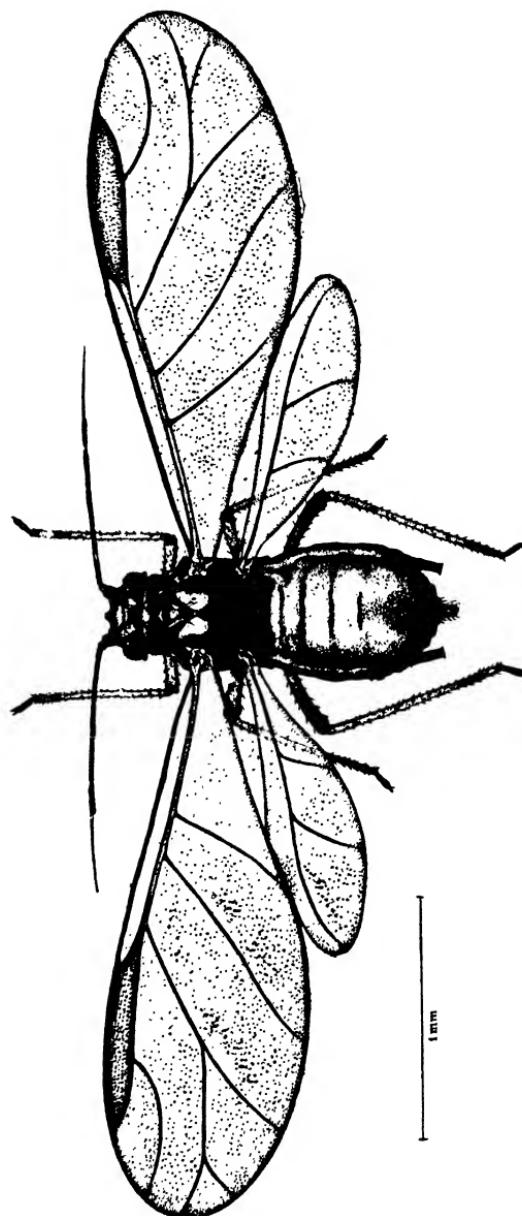


FIG. 93. *Aphis gossypii* Glover, alate viviparous female.

papillæ small; on last segment two large papillæ and a small one each side with a marked hair; four small dorsal apical papillæ. A few cephalic hairs. Short hairs scattered over body.

Length, 1·7 to 2 mm. (Theobald, 52).

Food Plants. Bulbs of *Tulipa*, *Gladiolus*, *Lilium*, *Scilla*, *Chionodoxa*, *Crocus*, *Iris*, also carrot and parsley roots.

Life History

Anuraphis tulipæ occurs upon the stored bulbs of tulips, gladioli, etc.; the insects commence to breed about March and continue until July, when they become winged and leave the bulbs. What is apparently the same insect feeds upon the roots of carrots and parsley, causing them to split.

Virus Transmitted by Anuraphis tulipæ

Tulipa Virus 1, causing "break" of tulips.

Geographical Distribution. British Isles, Germany, Wellington, New Zealand, United States of America, Bermuda.

Aphididæ (*continued*)

Aphis gossypii Glover. Melon aphid, cotton aphid.

DESCRIPTION

Alate Viviparous Female (see Fig. 93). Yellow, pale yellowish-green to dark green. Head, pronotum and thorax dark; a pale band before and behind pronotum. Abdomen with four black lateral spots, in some only three are visible, there may also be some dark dorsal spots making two or three broken transverse bands; in some forms the base of the abdomen may be orange, especially in dark forms. Antennæ shorter than body, varying from black to yellow, except at base and apex, where they are black; segment I. larger than II.; III. longer than IV. and nearly as long as VI. with five to eight round sensoria in a line; IV. and V. about equal; basal area of VI. about half V.; flagellum about three times as long as basal area. Eyes dark, large. Rostrum thin, yellow, apex dark, reaching to or just past second coxae. Cornicles black, cylindrical, in some slightly expanding basally; imbricate; about as long as segment IV. of antennæ, but thicker. Cauda green to almost black, about half the length of the cornicles, several long fine hairs. Anal plate dark. In some the cauda is almost yellow. Legs rather long and thin; apices of femora, tibiae and the tarsi dusky; in some the femora are all dark except at the base. Wing insertions yellow; stigma yellow, yellow-green to grey; veins thin, pale brown or black.

Length, 1·2 to 1·9 mm.

Apterous Viviparous Female (see Fig. 94). Colour variable, yellow, yellow-green, green of various deep shades, a few almost black.

Antennæ reaching to or beyond middle of body ; segments I., II. and VI. and apex of V. dark, rest yellow ; I. wider than II. ; III. longer than IV., not so long as VI., this latter seeming to vary in length to some extent ; IV. a little longer to nearly the same length as V. ; basal area of VI. more than half V. and about a quarter of flagellum. Eyes black ; rostrum yellow ; apex dusky in some, reaching often to third

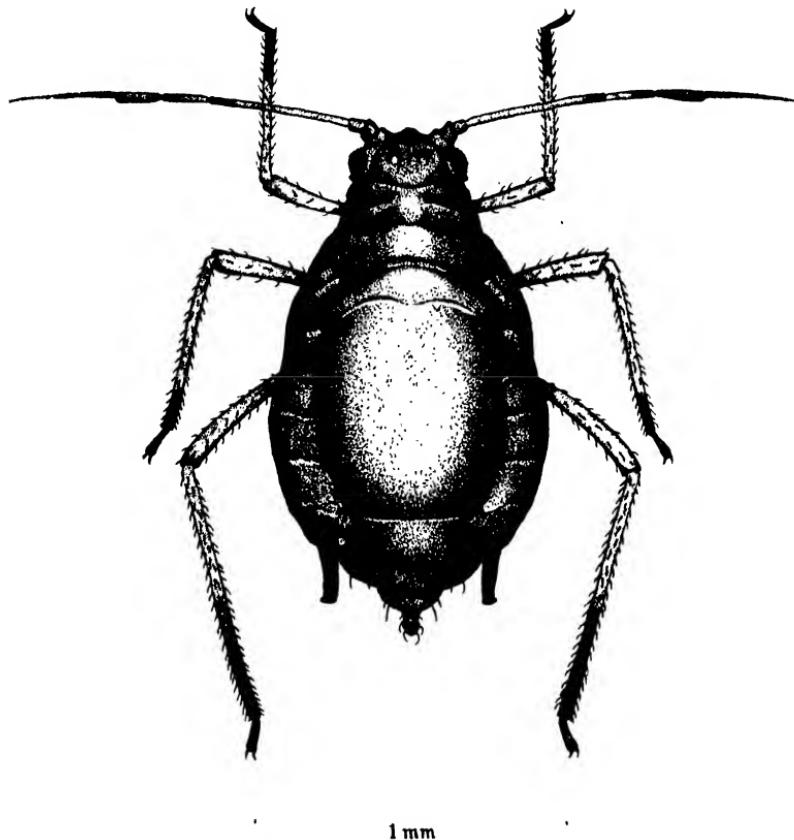


FIG. 94. *Aphis gossypii* Glover, apterous female.

coxæ. Cornicles black, olive green or green, as long as or even longer than antennal segment III., thick, cylindrical, broadened basally, divergent, imbricate. Cauda greenish to almost black, about half the cornicles ; three pale hairs each side, curved apically. Anal plate dark, hairy. A small conical papilla on each side of pronotum, one between cornicles and cauda and smaller ones cephalad. Legs yellow to green ; apices of tibiae and the tarsi dark, in some also apices of femora. Whole body now and then more or less mealy.

Length, 1.5 to 1.9 mm. (Theobald, 52).

Food Plants. *Chenopodium album*, *Cucumis melo*, *C. sativus*, *Cucurbita maxima*, *C. pepo*, *Lilium* spp., *Solanum* spp., *Rumex* sp., *Gossypium* sp., *Begonia* sp., *Hydrangea* sp., *Hibiscus* sp.

Life History

Little seems to be known of the sexuales of *A. gossypii*; in the British Isles it lives almost wholly under glass upon melons, cucumbers and liliaceous plants. It is a world-wide species and is often harmful to cotton, melons, etc., in America and Africa.

Viruses Transmitted by *Aphis gossypii*

Brassica Virus 3, causing mosaic of cauliflowers.

Cucumis Virus 1, causing cucumber and lily mosaic.

Phaseolus Virus 1, causing common bean mosaic.

Apium Virus 2, causing calico disease of celery.

Lilium Virus 1, causing rosette disease of lilies.

Allium Virus 1, causing yellow dwarf disease of onions.

Geographical Distribution. The British Isles and Europe generally, Russia, North America, Mexico, Brazil, Montserrat, B.W.I., Jamaica, Australia, Cape Colony, Transvaal, British East Africa, Uganda, Southern Nigeria, Bermuda, Sudan, Cairo, India, Malaya, China, Sumatra, Formosa, Fiji, British Guiana, Cyprus, Barbados, Queensland.

Aphididæ (continued)

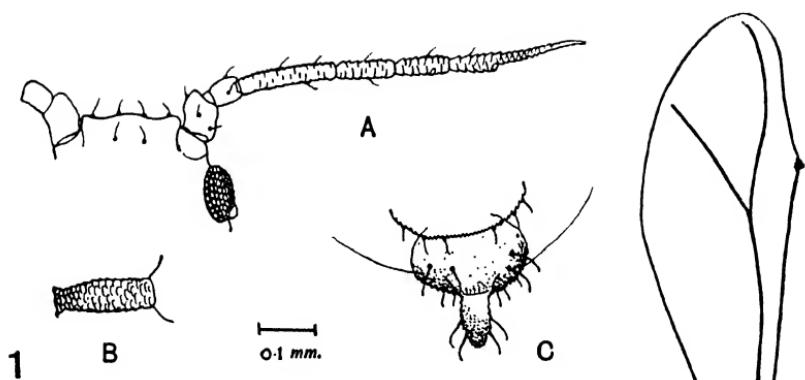
Aphis laburni Kalt. (*Aphis leguminosa* Theobald).

DESCRIPTION

Alate Viviparous Female. Black; shiny; antennæ black; III. to V. pale, brown, dull yellow to almost black; III. and IV. often almost white. Cornicles and cauda black. Legs black, tibiae dull white to dirty yellow; femora base pale. Antennæ about half the length of body; I. larger than II.; III. a little longer than IV., three to six sensoria, usually in middle, if six along whole length; IV. a little longer than V.; base of VI. about half V. Cornicles, black, long, cylindrical, as long as or a little longer than III. Cauda black, prominent, narrow, as thick as or a very little thicker than cornicles and almost half their length, with three to four hairs each side and two to three sub-apical curved ones. Tibiae slightly hirsute. Wings normal, somewhat tinted and very shiny; stigma brownish, insertions and some veins yellowish-green. A marked abdominal papilla between cornicles and cauda.

Length, 1·6 to 2 mm.

Apterous Viviparous Female. Black, with dull bluish-white meal or shiny. Antennæ black; segments III. and IV. and base of V. pale.



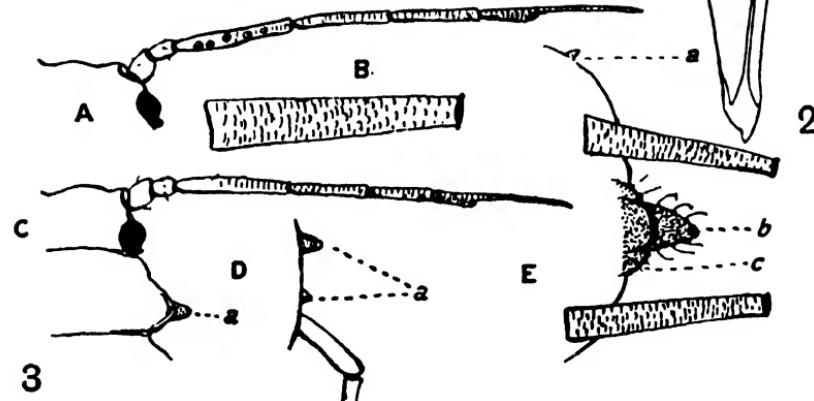
1

B

0.1 mm.

C

A



3

A.

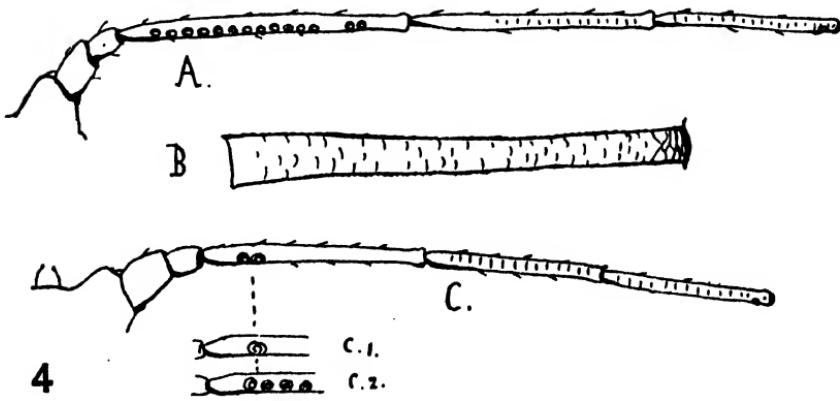
B

C.

4

C. I.

C. 2.



Cornicles and cauda black. Legs black, except tibiæ, which are pale with dark apices; femora with a small basal pale area on fore and mid legs and a large one on the posterior pair. Antennæ shorter than body; segment I. black, larger than II., which is also dark; III. longer than IV.; IV. and V. about equal; VI. not quite so long as IV. plus V., its basal area more than half length of V. and about half of flagellum. Cornicles thick, cylindrical, slightly expanding basally, pointing outwards, imbricate, longer than antennal segment III. Cauda long, narrow, about half length of cornicles and scarcely thicker, with four long hairs each side, very spinose. A prominent papilla each side of pronotum and a larger one between mid and hind legs, a smaller one between cauda and cornicles (see Fig. 95, 3).

Length, 1·5 to 1·9 mm. (Theobald, 52).

Food Plants. *Cytisus laburnum*, *Spartium scorarium*, *S. junceum*, *Genista* sp., *Indigofera*, *Vigna sinensis*, *Phaseolus lunatus*, *Acacia*, *Melilotus*, *Medicago*, *Poterium officinale*, *Deutzia scabra*, *Hibiscus syriacus*, *Zelkova acuminata*, and many other plants.

Life History and Habits

A marked, dark species which clusters on the leaves, shoots and pods of the laburnum, genista and broom, and often occurs in vast numbers on the laburnum, destroying the foliage and distorting the seed pods. In the British Isles it is common on broom in May and June and found on both laburnum and broom in August. The alatae fly from these to various leguminous plants. This aphid has been confused with *A. rumicis*, from which it can be differentiated by the fewer sensoria on antennal segment III. of the alate female.

FIG. 95.

1. *Aphis maidis* Fitch, apterous female.
 - A. Head and antenna.
 - B. Cornicle.
 - C. Cauda.
2. Hindwing of *Hysteroneura setaria* Thos.
3. *Aphis laburni* (leguminosæ Theob.).
 - A. Head and antenna of alate female.
 - B. Cornicle.
 - C. Head and antenna of apterous female.
 - D. Lateral abdominal tubercles.
 - E. (a) abdominal papilla, (b) cauda, (c) anal plate and cornicles.
4. *Macrosiphum pelargonii* Kalt., alate female.
 - A. Antenna.
 - B. Cornicle, apterous female.
 - C. Antenna.
 - C1 and C2. Variations in sensoria. (3 & 4 after Theobald)

Virus Transmitted by *Aphis laburni*

Arachis Virus 1, causing rosette disease of groundnuts.

Geographical Distribution. British Isles, Germany, Belgium, Italy, America, Malaya, South Africa, India, Ceylon, Formosa, Japan, Egypt, Sumatra, Java.

Aphididæ (*continued*)

Aphis maidis Fitch. The corn leaf aphid; the sugar cane aphid.

DESCRIPTION

Alate Viviparous Female. Head black. Antennæ black, and with six segments, or seven if the filament be counted. Antennal sensoria circular, sixteen to twenty on III., four on IV., several at apical end of V., and also at apical end of the basal part of VI. Eyes dark brown or black. Thorax and legs black. Abdomen pale bluish-green. Three black spots on each side of the body and anterior to the cornicles, and a black basal spot surrounding each cornicle. Posterior to the cornicles are two black spots, one on each side, and also three more or less distinct transverse black bands. Cornicles black, slightly incrassate at middle, dilated at apex. Distal half of dorsally curved style black, and the remainder margined with black to the base.

Measurements: Length of body, 1.709 mm.; width, 0.618 mm.; wing expanse, 5.786 mm.; antennæ, I., 0.065 mm.; II., 0.057 mm.; III., 0.293 mm.; IV., 0.154 mm.; V., 0.154 mm.; VI., basal, 0.106 mm.; filament, 0.228 mm.; total, 1.057 mm.; cornicles, 0.130 mm.; style, 0.081 mm. (12).

Apterous Viviparous Female. Head black. Antennæ black excepting third segment. Eyes very dark reddish-brown. Rostrum dark, its apex black, shading to brown. General colour of body blue-green. Fore segments and tip of abdomen very dark green. Legs black, excepting middle portion of femur. Cornicles black, slightly incrassate at the base and with a dark green basal patch. The adult gradually becomes darker in colour as it grows older until it is almost black with a slightly greenish or brownish tint (see Fig. 95, 1).

Measurements: Length of body, 2.363 mm.; width, 1.091 mm.; antennæ, I., 0.067 mm.; II., 0.054 mm.; III., 0.193 mm.; IV., 0.115 mm.; V., 0.111 mm.; VI., basal, 0.077 mm.; filament, 0.176 mm.; total, 0.793 mm.; cornicles, 0.203 mm.; style, 0.101 mm. (12).

Food Plants. Though the usual food plants are maize (corn), sugar cane, sorghum and broom corn, *A. maidis* feeds also on various other plants such as barley, onion, *Setaria glauca* and *Oxalis*. It has also been observed by Davis breeding on *Panicum crus-galli* and *P. sanguinale*.

Life History

The bionomics of *Aphis maidis* in the United States are not

fully understood, as it is not yet known how this aphid overwinters. In Illinois it first appears in midsummer on corn and broom corn and continues to reproduce pathogenetically until the autumn, when it disappears from the fields. The most likely explanation is that there exists an alternate food plant on which the insect overwinters.

Viruses Transmitted by *Aphis maidis*

Allium Virus 1, causing yellow dwarf disease of onions.

Saccharum Virus 1, causing mosaic disease of sugar cane.

Geographical Distribution. This aphid has a very wide distribution, being found in all parts of the United States where corn (maize) is grown, that is, from Maine to California and Texas. It has also been recorded from Australia, Japan and the principal sugar cane-growing countries.

Aphididæ (continued)

Aphis rhamni Boyer (*A. rhamni* Koch., *A. abbreviata* Patch). The buckthorn aphid.

DESCRIPTION

Alate Viviparous Female. Green; head and thoracic lobes black; a green band on pronotum or all green; black lateral abdominal spots; black cauda and cornicles; antennæ deep brown, base of III. paler; legs yellowish-green, apices of femoræ, tibialæ and the tarsi dark. Eyes black. Antennæ shorter than body; segment I. a little wider than II.; III. longer than IV., about as long as flagellum, with ten to fourteen round sensoria mainly along one side up to apex; IV. equal to V. or a little longer, with three to six sensoria; V. with one to three plus one and not quite twice base of VI.; flagellum three times base. Rostrum to second coxæ. Cornicles not quite, or about, as long as antennal segment V.; cylindrical and imbricate, variable in length; cauda acuminate, not quite so long as cornicles, with a few long, pale, curved hairs. Abdominal lateral papillæ, pale, small; each segment with a few median irregular rows of short hairs; three large spots on each side and a dark patch at base of cornicles caudad, a dark area between cornicles and cauda and some small dusky glandular areas on body. Wings and legs normal.

Length, 1·4 to 1·7 mm.

Apterous Viviparous Female. Pale green to pale yellowish-green with some deeper green lines; antennæ same colour as body apices, dusky; cornicles and cauda green or yellow-green, in some the cornicles slightly dusky at apices; legs same colour as body, apices of femora and the tarsi dusky. Antennæ shorter than body; I. larger than II.; III. a little longer than IV., but now and then equal to it; IV. slightly longer than V., now and then equal to it, so that III., IV. and V. are

sometimes equal; flagellum about two and a half times base; segments with a few short hairs. Rostrum reaches to between second and third coxae, sometimes to the third. Cornicles cylindrical, about as long as antennal segment III., rather thick. Cauda rather thick and blunt, a

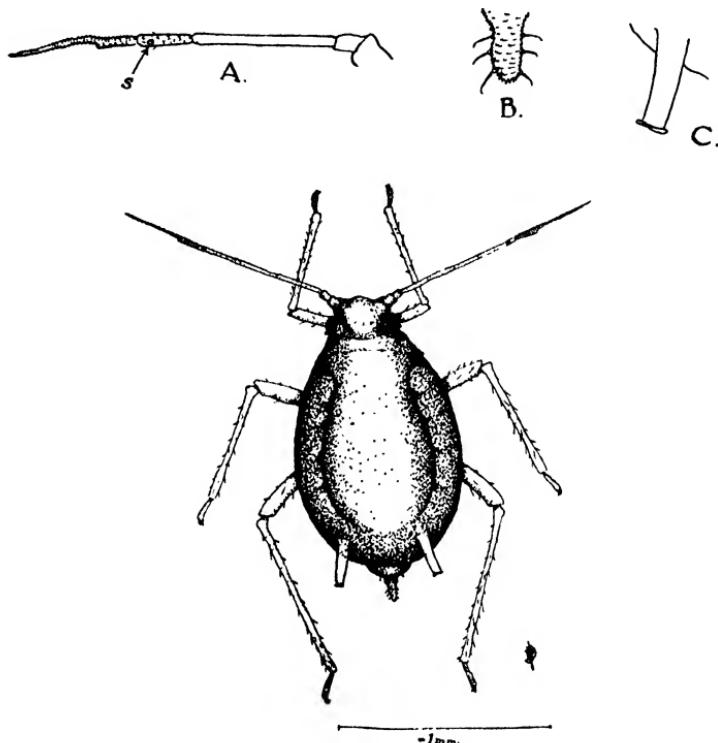


FIG. 96. *Aphis rhamni* Boyer (= *A. solanina* Pass. and *A. abbreviata* Patch) apterous viviparous female. $\times 27$.

- A. Antenna.
- B. Cauda.
- C. Cornicle. all $\times 60$.
- S. Sensorium : note that this sensorium is usually found on the fifth antennal segment.

little more than half cornicles, three hairs each side and one or two dorso-apical ones. Lateral abdominal papillæ small. A few hairs on body. Legs with many spine-like hairs on tibiae (see Fig. 96).

Length, 1 to 1.2 mm. (Theobald, 52).

Food Plants. Buckthorns, *Rhamnus catharticus* and *R. frangula*, potato and many other plants in America. It is nearly always present in small colonies on potatoes in England. Some of the

secondary host plants in America are as follows : *Chenopodium*, *Alisma*, *Beta*, *Stellaria*, *Berberis*, *Brassica oleracea*, *Oxalis*, *Fragaria*, *Impatiens*, *Malva*, *Viola*, *Pastinaca*, *Galeopsis*, *Plantago*, etc.

Life History and Habits

In England this aphid is to be found on buckthorns in May, June and July, migration then taking place to other secondary hosts, of which the potato plant is one. It also occurs on the potato during June. The sexuales occur on buckthorn in October and November, and the eggs are laid in axils of the buds.

Virus Transmitted by *Aphis rhamni*

Solanum Virus 3, causing mottle or leaf-drop in potatoes.

Geographical Distribution. Widely distributed in the British Isles ; it has been recorded from Brussels, and it also occurs in America.

Aphididæ (continued)

Aphis rubicola Oestl. (*rubiphila* Patch). The small raspberry aphid (U.S.A.).

DESCRIPTION

An aphid about the size of *gossypii* and resembling it in wing venation and length of cornicle. The alate female has head shiny black, antenna dusky, eyes black, prothorax with pale membrane and prominent lateral tubercles ; thorax shiny, wings with slender dark brown veins ; abdomen glabrous, pale green, lateral tubercles prominent, cornicles nearly concolorous, perhaps a little darker, cauda green and hairy. The sensoria of antennal III. are few, four to five, and confined chiefly to the distal two-thirds (see Fig. 92, 4).

The apterous female is pale green with slightly pulverulent abdomen. Cauda and cornicles lighter than abdomen, with the tips of the latter black (Patch).

An important point in identification lies in the shape of cauda and the number and position of the hairs.

Food Plants. The cultivated red, purple and black raspberries.

Life History and Habits

The eggs are laid in the axils of the leaves near the tip of the cane. They are usually laid on the cane in the crevice formed by the closely appressed bud which grows into a fruiting lateral in the following season. The stem-mothers hatch from the eggs when the buds are showing the first green, and feed on the outer surface of the folded leaves. The stem-mothers are dark bluish-green,

plump, and larger than the adults of the succeeding generations. They are somewhat powdery on the upper surface, and the legs, antennæ, head and cornicles are grey. As they reach maturity they become lighter green and the appendages lose their grey colour and become brown. By about the end of the first week in May, second generation young are abundant on laterals, which by this time are about 4 to 7 inches long. Usually the second generation, and probably also the third and fourth, are very abundant. They multiply on the new succulent suckers in June, but rarely thrive on the fruiting laterals. In Ontario winged forms are very unusual in the open. The summer generations are entirely colourless and very small. They feed along the veins on the under sides of the leaves. Apterous sexual forms appear in November and commence egg-laying. The majority of the eggs are laid near the tip of the cane of red varieties, and, since this is usually pruned off before they hatch, a large proportion of the eggs are removed.

It has been suggested that this aphid thrives better on the new suckers affected with mosaic than on new healthy suckers (40).

Viruses Transmitted by *Aphis rubicola* Oestl.

Rubus Viruses 3 and 3A, causing raspberry leaf-curl (alpha and beta).

Geographical Distribution. *Aphis rubicola* was described for the first time by Patch, in 1914, from Maine, U.S.A. It seems fairly common in North America and parts of Canada, but does not occur in the British Isles.

Aphididæ (continued)

Aphis rumicis (*Aphis fabæ* Scop.) Linn. The bean aphid; "black-fly."

DESCRIPTION

Apterous Viviparous Female (see Fig. 97, 3). Body elongate oval, colour variable, black to olive green, often with irregular darker pigmented areas over the abdomen, small hairs scattered over the body. Head, eyes black with prominent accessory eyes; antennæ six-jointed, segment I., apical portion of segment V. and proximal portion of segment VI. black, remainder paler, a single sub-apical sensorium on segment V., a compound sensorium on segment VI. Thorax with a prominent prothoracic tubercle on each side; legs black, tibiæ and proximal portion of femora paler; segments bearing stout hairs, especially the tibiæ. Abdomen with two lateral tubercles on each side, and sometimes one or two small indefinite tubercles; cornicles black, tubular, imbricated, slightly tapering distally, usually one and one-third to one

and a half times the length of the cauda ; cauda with distal half slightly spoon-shaped, black, clothed with short stout bristles and several long curved hairs.

Length, 2·5 mm.

Alate Viviparous Female. Body, head and thorax black to brownish-black, abdomen varying from brownish-black to olive green, usually with irregular darker pigmented areas, small hairs scattered over the body. Head black, eyes black with prominent accessory eyes, antennae dirty brown to black, varying in length, about two-thirds length of body, rostrum dark towards distal end. Thorax with two prominent lateral tubercles on prothorax ; wings normal ; legs somewhat longer than in the apterous viviparous female, otherwise similar. Abdomen varying in colour from dark velvet black to olive green, usually with five irregular pigmented areas along the lateral dorsal area and irregular transverse areas segmentally arranged, lateral tubercles prominent ; cornicles black, tubular and slightly tapering ; cauda not so large as in the apterous viviparous female.

Length, 2·4 mm. (8).

Food Plants. The host plants are very numerous, and a complete list cannot be given here. Some of the more important are as follows : *Papaver* spp., *Rumex* spp., *Chenopodium* spp., *Beta vulgaris*, *B. maritima*, *Atriplex* spp., *Euonymus europaeus*, *Vicia faba*, *Vicia* spp., *Phaseolus vulgaris*, *Allium cepa*, *Digitalis purpurea*, *Carduus* spp., *Humulus lupulus*, *Solanum* spp., *Heraclium sphondylium*, spinach, turnips, rhubarb, *Tropaeolum*.

Life History

This aphid winters in the egg stage as a rule, the ova being laid in autumn on the *Euonymus* or *Rumex*, rarely on *Viburnum opulus*, and in America on *Chenopodium*. The eggs are laid indiscriminately, some on young wood, most at the base of buds. They are at first dull yellow, but soon become black. The ova hatch in spring and give rise to the stem-mothers, which produce apterous viviparae, and by June nymphæ occur ; these soon hatch into alatae, and during the latter month and till July the winged forms migrate to a host of herbaceous plants, particularly beans, poppies and mangolds. Those that have wintered on docks (*Rumex*) also breed rapidly on those plants, and some assume wings and migrate to other herbaceous plants, while others continue to reproduce in small numbers on the dock throughout the summer. Late in summer alatae appear on the herbaceous plants, these return migrants produce egg-laying females on the *Euonymus* and *Viburnum*, later alate males develop and fly and join the apterous oviparae and fertilise them (Theobald, 52).

Viruses Transmitted by *Aphis rumicis*

Beta Virus 2, causing mosaic of sugar beet.

Phaseolus Virus 1, causing common bean mosaic.

Pisum Virus 2, causing common pea mosaic.

Trifolium Virus 1, causing mosaic of white clover.

Allium Virus 1, causing yellow dwarf disease of onions.

Geographical Distribution. Widely distributed throughout Europe generally, Africa, India, Formosa, Japan, United States and Canada, Brazil, Sakhalin. According to Hall it is not common in Egypt.

Aphididæ (continued)

Brevicoryne brassicæ Linn. (*Aphis brassicæ* Linn.). Mealy cabbage aphid.

DESCRIPTION

Alate Viviparous Female. Head black, pronotum deep brown, rest of thorax black. Abdomen yellowish-green to dull green, a row of four black spots on each side and dark transverse bars in centre, more or less broken. Cornicles dark, short. Antennæ about as long as body, dark brown. Cauda dark. Legs brown to almost black. Antennæ with segment I. larger than II. ; III. two and a half to three times length of IV., with fifty to sixty sensoria over whole length, not so long as VI. ; IV. about as long as or a little longer than V. ; VI. with basal area more than half of V. ; flagellum three times as long as basal area. Rostrum pale at base, apex dark, reaching second coxæ. Cornicles short, not quite so long as basal area of antennal segment VI., swollen in middle, often much constricted at base ; faintly imbricate. Cauda slightly longer than cornicles to about same length, triangulate, three hairs each side and one dorso-apical. Anal plate dark, more or less quadrilateral, with a few hairs. Legs rather long and thin ; tibæ hairy ; femora pale at base. Eyes large, black. Veins brown ; stigma paler greyish-brown ; insertions green.

Length, 1·9 to 2 mm.

Apterous Viviparous Female. Greyish-green, densely covered with a fine white mealy powder, eight black transverse spots on each side, variable in size and form, sometimes broken in middle so as to form four spots ; smaller black lateral spots ; posteriorly these median black spots may fuse and form bars. Antennæ shorter than body, dark, except greater part of segment III. ; I. a little larger than II. ; III. about three times as long as IV. ; IV. and V. about equal, in a few IV. may be shorter ; VI. longer than IV. plus V. ; basal area about one-quarter of flagellum. Rostrum green at base, apex black, reaching to second coxæ, rather thin. Cornicles dark, short, narrowed at apices and in some at base ; faintly imbricate, not quite so long as antennal segment V. Cauda about as long as cornicles, bluntly triangulate ;

three hairs each side. Anal plate dark, more or less quadrilateral. Legs dark, almost black; base of femora paler; tibiae hairy. Eyes black.

Length, 2·1 to 2·4 mm. (Theobald, 52).

Food Plants. All species of *Brassica*, *Capsella bursa-pastoris*, charlock (*Sinapis arvensis*), field cress (*Isatis tinctoria*), *Diplotaxis tenuifolia*, radish (*Raphanus sativus*), *Bunias erucago*, *Erysimum canescens*, *Crambe maritima*, *Lepidium sativum*, *Thalictrum minus*, *Spinacea oleraceus*.

Life History and Habits

Theobald (52) describes the life history as follows: The aphis appears first in May, usually starting in small clusters beneath the leaves, and by June the alatae appear and fly far and wide. Several generations occur, and the insects may be found on the plants right into December. In November the alate males and oviparous females appear, and the latter lay their eggs on the winter greens. The black eggs often occur in great masses. The stem-mother appears, hatched from the winter eggs in March. This form will live for over a month and at first reproduces very slowly. The adults derived from these live about the same time, and as the summer goes on increase more and more rapidly; the greatest rate of increase is usually in September and October and into the early part of November. Sexual forms may occur as early as October, but most in November and December. The ova seem to be laid indiscriminately beneath the leaves and on the stems, now and then they may be found on the upper surfaces. According to Petherbridge and Mellor (37) the life history varies from season to season and may differ slightly from that given above. Thus, in 1934, the aphis overwintered in the egg stage only on cultivated cruciferous crops, particularly brussels sprouts, whereas observations strongly suggest that in 1935 it overwintered both as viviparous females and in the egg stage. The hatching of the eggs was much later in 1934 than in 1935, but the first winged forms were produced at about the same date.

Viruses Transmitted by *Brevicoryne brassicæ*

Brassica Virus 2, causing mosaic of Brassicas and stocks.

Brassica Virus 3, causing mosaic disease of cauliflowers.

Brassica Virus 4, causing mosaic disease of cruciferous plants.

Matthiola Virus 1, causing mosaic disease of stocks.

Phaseolus Virus 1, causing common mosaic of bean.

Allium Virus 1, causing yellow dwarf disease of onions.

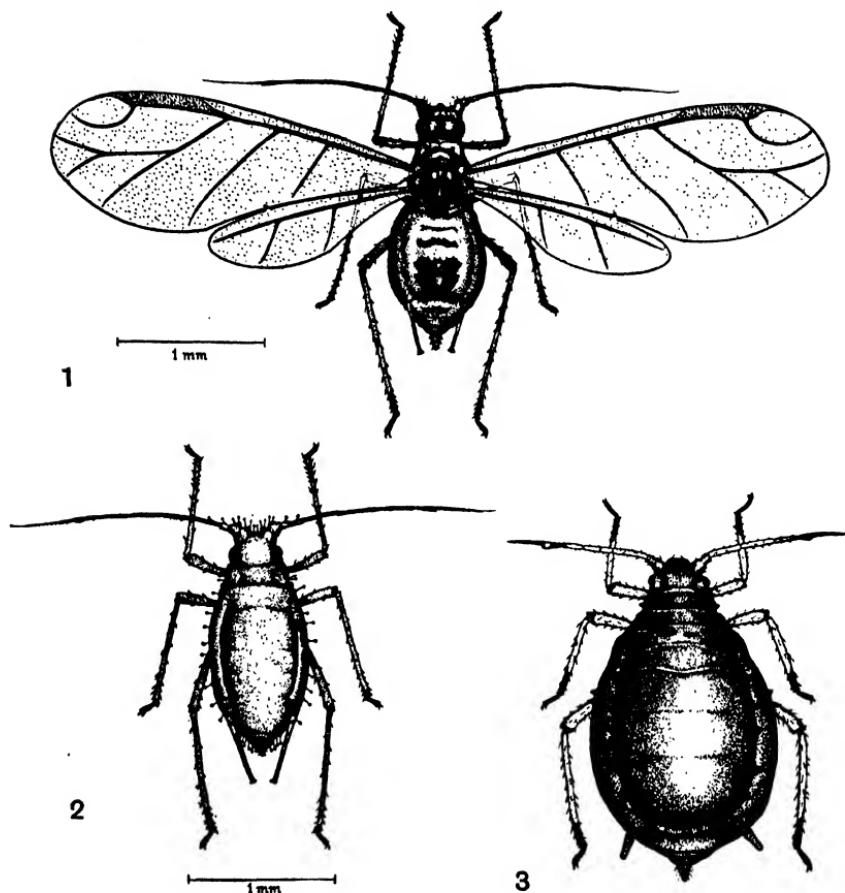


FIG. 97.

1. *Capitophorus fragæfolii* Ckll., alate female.
2. *Capitophorus fragæfolii* Ckll., apterous female.
3. *Aphis rumicis* L., apterous female.

Geographical Distribution. This aphis is widely distributed ; it occurs all over Great Britain, Europe, America, British Columbia, Japan, India, Africa and Formosa.

Aphididæ (continued)

Capitophorus fragæfolii Cockll. (*C. fragariae* Theob., *C. potentillæ* Wlk., *Myzus fragæfolii* Cockll.). The delicate strawberry aphis, the American strawberry aphis.

DESCRIPTION

Alate Viviparous Female (see Fig. 97, 1). Head dark brown. Antennæ dark brown. Pronotum pale ; yellowish-green. Mesonotum with dark brown median area. Abdomen pale yellowish-green with a dusky patch on the apical half and a few small dusky spots on the basal segments and dusky lateral spots. Legs pale yellowish-green ; apices of femora and tibiae dusky ; tarsi dark. Cornicles pale yellowish-green. Wings with smoky black veins and smoky black stigma. Hairs capitate, but rather scanty ; four on front of head, some on the two basal antennal segments and short ones on the body, the heads being slightly swollen. Antennal segment III. with thirty-two to thirty-six sensoria disposed all along it ; IV. with five to eight sensoria ; III. about as long as IV. plus V. ; VI. a little longer than IV. and V. Cornicles show faint imbrication, not projecting beyond pale green cauda, which has two pairs of lateral hairs and one median sub-apical one.

Length, 2 to 2·5 mm.

Apterous Viviparous Female (see Fig. 97, 2). Very pale green, often semi-transparent. Head, thorax and abdomen with capitate hairs. Antennæ as long as body ; segment I. rather large with a thick, blunt swelling on inner side ; II. small ; III. long, not quite so long as IV. plus V., which are equal ; VI. longer than IV. plus V. ; I. with several, II. with two large and some quite small capitate hairs ; III. with one or two on the inside. Cornicles long and thin, almost transparent, with faint imbrication ; projecting some distance beyond the cauda. Cauda pallid green, with pairs of lateral hairs and a median sub-apical one. Legs pallid green, almost transparent ; tarsi slightly dusky ; femora and tibiae with capitate hairs. Eyes blackish (Theobald, 52).

Length, 1 to 1·5 mm.

Food Plants. *Centranthus ruber*, red valerian ; *Fragaria vesca*, cultivated strawberry.

Life History

Very little is known of the habits of this aphid ; it can be induced to form colonies on various host plants under glasshouse conditions. Among these the wild strawberry, the strawberry-leaved *Potentilla* (*Potentilla fragariastrum* Ehrh.) and the silver-weed (*P. anserina* L.) are most preferred. Under natural conditions the aphid has only been observed on the cultivated strawberry. The egg stage is not known (38).

Viruses Transmitted by *Capitophorus fragæfolii*

Fragaria Virus 1, causing yellow edge disease of strawberries.

Fragaria Virus 2, causing crinkle disease of strawberries.

Fragaria Virus 3, causing witch's broom disease of strawberries.

Geographical Distribution. Fairly widespread in the British

Isles, especially in the strawberry-growing districts. It appears also to be the common strawberry aphid of the United States of America.

Aphididæ (continued)

Hysteroneura (Aphis) setariæ Thos. The rusty plum aphid.

DESCRIPTION (after Gillette)

Vertex almost flat in apteræ, distinctly convex in alatae. Frontal tubercles slightly developed, equalling vertex in alatae, exceeding vertex in apteræ. Secondary sensoria circular, slightly convex, arranged in rather irregular rows along almost the entire length of joint, fifteen to twenty-one on III., three to seven on IV., none on V.; absent in apteræ. Hairs on vertex, antennæ and tibiæ, 0·01 to 0·015 mm. long, pointed, fine and sparse. Cornicles slightly tapering, with indistinct flange, imbrications close and crenulate. Cauda slender, cylindrical, somewhat wider proximally, bearing two pairs of lateral hairs. Lateral tubercles present on prothorax and abdominal segments I. to V. and VI. and VII. Forewings with second fork of media about 0·33 of the distance from margin of wing to first fork. Hindwings with cubitus vestigial or absent. Oviparae with hind tibiæ not noticeably swollen and bearing about nineteen sensoria mostly in middle portion.

Males apterous.

Colour, all forms: Brown, dark in apteræ, lighter in alatae; cauda and legs white; cornicles black.

Measurements: Apterous summer viviparae—body to base of cauda, 1·4 to 2·3 mm.; hind tibiæ, 0·80 to 1·1 mm.; hind tarsi, 0·11 to 0·13 mm.; antennæ, 1·4 to 1·8 mm.; III., 0·28 to 0·43 mm.; IV., 0·20 to 0·27 mm.; V., 0·16 to 0·25 mm.; VI., 0·10 to 0·11 mm.; cornicles, 0·25 to 0·40 mm.; rostrum obtuse, joint IV. plus V. broad, 0·11 mm., attaining or surpassing second coxae.

Alate viviparae—same as apterae, except that rostrum hardly attains second coxae.

The appearance of this insect is typical of the genus "Aphis" with the exception of the venation of the hindwings, which have but a single cross vein (see Fig. 95, 2). The filament of antennal segment VI. is quite long, being six to eight times the length of the base of this segment.

Food Plants. Maize (corn), sugar cane, *Eragrostis* sp., *Sorghastrum nutans*, *Panicum capillare*, *Paspalum dilatatum*, and Bermuda grass (*Cynodon dactylon*).

Virus Transmitted by *Hysteroneura setariæ*

Saccharum Virus 1, causing sugar cane mosaic.

Geographical Distribution. From various states in the U.S.A., Florida, Illinois, Indiana, Iowa, Kansas, Louisiana, Missouri, New York, Oklahoma, South Carolina, Texas, Wisconsin.

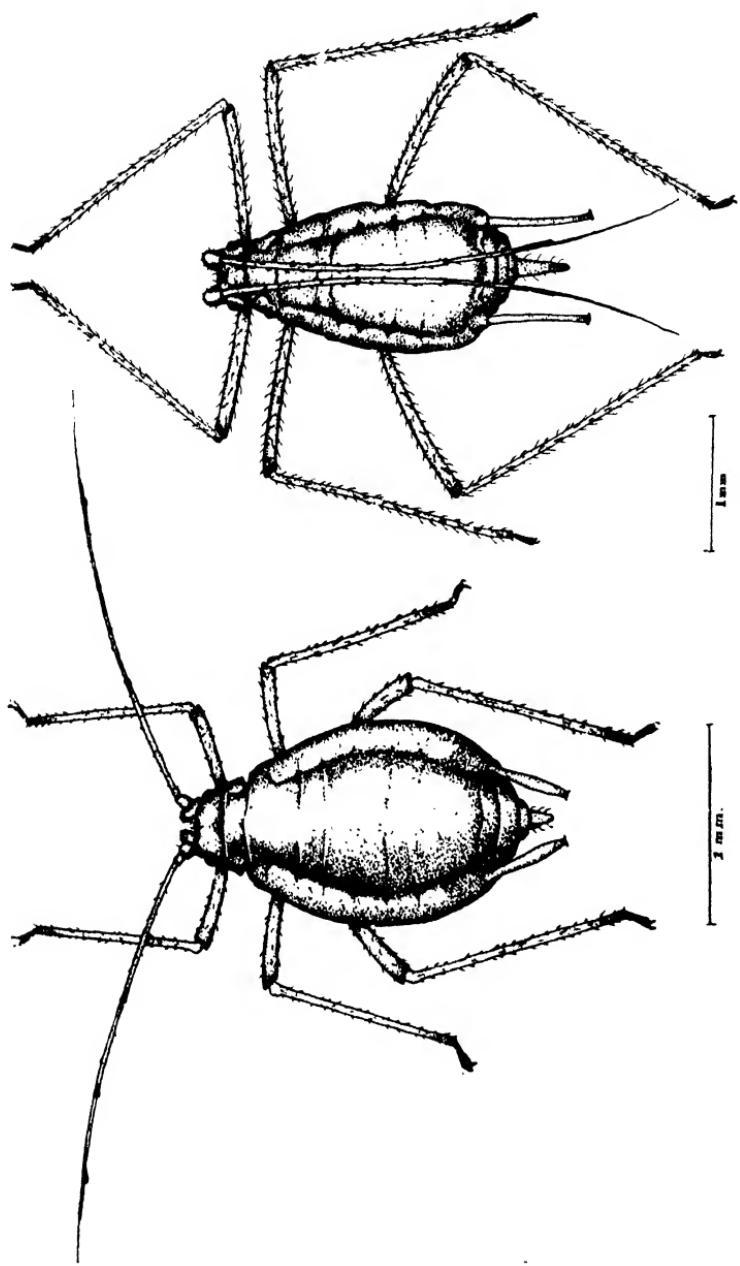


FIG. 98.
Left. *Myzus persicæ* Sulz., apterous viviparous female.
Right. *Macrosiphum gei* Koch, apterous viviparous female.

Aphididæ (continued)

Macrosiphum gei Koch. (*solanifolii* Ashm.). The pink and green potato and rose aphid.

DESCRIPTION

Alate Viviparous Female. Head somewhat narrow with many finely capitate hairs ; eyes black ; ocelli well developed ; antennæ with the two basal segments pale green, remainder dark or fuscous, as long as or longer than the body ; segment III. with about fourteen sensoria in a row along its internal face, extending for about three-fourths the length of the segment ; several small faintly capitate hairs on all segments. Wings well defined, dark veins and greenish-brown stigma. Legs, coxae and trochanters green, femora and tibiae somewhat darker green and darker at distal ends, tarsi dark. Abdomen elongate with rows of short hairs on all segments ; cauda not quite half the length of cornicles, pale green, ensiform, finely spinose and bearing several lateral hairs, and one or two sub-apical median hairs ; anal plate rounded, spinose and bearing a few long hairs ; cornicles yellowish-green basally, darker on distal portion, slender, tapering distally, reticulate at distal end for about one-fifth of its length and the surface sculptured over remaining portion.

Length, 2.9 to 3.3 mm. (9).

Apterous Viviparous Female (see Fig. 98). Body green to yellowish-green, more or less uniformly yellowish-green on the venter. Head green, bearing a few short hairs ; eyes black ; rostrum yellowish-green, darker at distal end, reaching to coxae II. ; antennæ as long as or longer than the body, fuscous, apex of segments darker and also the whole of segment VI. ; segment III. with a few (about three) round sensoria on inner face near its base, all segments with a few faintly capitate hairs. Legs, yellowish-green with dark tarsi and distal end of tibiae and femora dark. Abdomen elongate with rows of short hairs on all segments ; cauda slightly less than half the length of cornicles, pale green, ensiform, finely spinose, bearing several lateral hairs and one or two sub-apical median hairs ; anal plate as in the winged viviparous female ; cornicles pale green basally, darker on distal portion, about as long as antennal segment III., reticulate at distal end for about one-fifth of the length.

Length, 4 to 4.1 mm. (9).

Food Plants. *Spiraea ulmaria*, *S. filipendula*, *Stellaria graminea*, *Geum urbanum*, *Epilobium montanum*, *E. angustifolium*, potato, roses ; also recorded from the following : *Zea mays*, *Iris* spp., *Gladiolus* sp., *Fagopyrum esculentum*, *Capsicum* sp., *Solanum melongena*, *Lamium album*, tomato, soya beans, *Atriplex* sp., *Chenopodium album*, *Amaranthus retroflexus*, *Brassica rapa*, *Capsella bursa-pastoris*, *Pyrus malus*, *Phaseolus vulgaris*, *Pisum sativum*, *Physalis* spp., *Solanum jasminoides*, *Aster* sp., *Lactuca* sp., *Cineraria* sp., *Sonchus oleraceus*, *Chrysanthemum* sp., *Belladonna*,

Humulus lupulinus, *Glaucium luteum*, yellow horned poppy, *Tropaeolum*, *Borago*, *Lilium*, *Veronica*, *Convolvulus*.

Life History

In the United States of America the life history is recorded by Patch as follows. The green and pink aphid of the potato is found in spring upon rose bushes, feeding on the succulent growth, and especially abundant near the flower buds. Wingless as well as winged forms may migrate. These pass to the potatoes, upon which they increase enormously. A single female may produce more than fifty young in two weeks, and in warm weather these become mature in two weeks. By mid-September the autumn migration is over and the aphides have deserted the potato fields. They may go to other plants, but the rose seems to be the favourite. The last generation of the year consists of wingless egg-laying females and alate males, which appear, in Maine, U.S.A., about September 21st. The rose is evidently the favourite over-wintering plant, and the aphides occur in colonies upon roses up to July, and by this time the summer generations may be found upon the potato. In this country there may be a similar sequence of movements, though the rose seems less frequented. In addition to the potato plant, upon which it is always present in the summer, it also occurs upon the iris, tulip and nettle. It is possible that it may also overwinter in the egg stage upon the potato, since it has been found feeding on the sprouting tubers in early spring together with *Myzus persicae* and *M. pseudosolani*.

Viruses Transmitted by Macrosiphum gei

Cucumis Virus 1, causing mosaic of cucumbers.

Phaseolus Virus 1, causing common mosaic of beans.

Phaseolus Virus 2, causing yellow mosaic of beans.

Pisum Virus 1, causing enation mosaic of peas.

Nicotiana Virus 1, causing mosaic of tobacco (from tomato only).

Solanum Virus 12, causing "spindle tuber" disease of potatoes.

Solanum Virus 13, causing unmottled curly dwarf disease of potatoes.

Tulipa Virus 1, causing "breaking" in tulips.

Allium Virus 1, causing yellow dwarf disease of onions.

Iris Virus 1, causing stripe or mosaic disease of irises.

Geographical Distribution. Common generally throughout the British Isles and probably Europe generally. It is also present

in Canada, Egypt and the United States of America, where it appears to be widely distributed.

Aphididæ (*continued*)

Macrosiphum pisi Kalt. Pea aphid, green pea louse.

DESCRIPTION

Alate Viviparous Female. Green; head sometimes yellowish; eyes red; antennæ very long, reaching beyond cauda, pale, segments I. and II. darker green, segment VI. black, segment III. with a row of ten to twenty sensoria; rostrum shorter than in the apterous viviparous female, wings clear, veins brownish, stigma yellowish; legs green, darkened as in the apterous viviparous female, cornicles long and thin, reaching beyond cauda, non-reticulate, apex darker, imbricated, cauda long and green.

Length, 2·70 mm.

Apterous Viviparous Female. Body green to pale green, shining, usually darkening towards posterior; eyes red or reddish-brown; antennæ long, arising from prominent tubercles; apices of segments dark, segment VI. dark brown and the longest, segment III. with one to three circular sensoria near the base; rostrum reaches as far as coxae of second pair of legs; legs long and slender, green, apices of femora and tibiae dusky, tarsi black; cornicles long and slender, broadest at base, non-reticulate; cauda green, long and ensiform with a few long hairs.

Length, 2·5 to 3 mm.

Food Plants. Mostly leguminous plants, cultivated peas, both culinary and ornamental, wild everlasting pea (*Lathyrus sylvestris*), red clover (*Trifolium pratense*), wild white clover (*T. repens*), alsike (*T. hybridum*), broad bean (*Vicia faba*); also lucerne (alfalfa), vetches, wild and cultivated broom (*Spartium*). Theobald also records it from greenweed (*Genista tinctoria*) and shepherd's purse (*Capsella bursa-pastoris*).

Life History

This aphid passes its life on peas, clovers, vetches, lucerne (alfalfa), etc. It has no true alternate host plant of a different family, such as is the case with many aphides. *M. pisi* usually appears on peas and beans in late June and July, and goes on breeding on them until August or late September, when the aphides become winged and fly off to clovers and perennial peas. It winters on such perennial Leguminosæ as the everlasting peas (*Lathyrus*) and clovers (*Trifolium*), etc., and at the same time may occur on such plants all the year round. It winters mainly in the egg stage, but apterous females may also overwinter. In summer

many become alate and fly from the clovers, etc., to field and garden peas and beans. In America, Davis records that it usually passes the winter on clovers either as eggs or oviparous females. Further north it may winter exclusively as ova, while further south, in Tennessee, the sexual forms are rare, and it usually winters as alate and apterous females (Theobald, 52).

Viruses Transmitted by *Macrosiphum pisi*

Phaseolus Virus 1, causing common mosaic of beans.

Phaseolus Virus 2, causing yellow mosaic of beans.

Pisum Virus 1, causing enation pea mosaic.

Pisum Virus 2, causing common mosaic of peas.

Trifolium Virus 1, causing mosaic of white clover.

Medicago Virus 1, causing common mosaic of lucerne (alfalfa).

Medicago Virus 2, causing mosaic of lucerne (alfalfa).

Allium Virus 1, causing yellow dwarf disease of onions.

Geographical Distribution. *M. pisi* is widely distributed ; it occurs in Europe, America, Africa, India and Japan.

Aphididæ (*continued*)

Macrosiphum pelargonii Kalt. (*Aphis pallida* Walk., *Siphonophora malvæ* Pass.).

DESCRIPTION

Alate Viviparous Female. Green ; head and thoracic lobes pale brown to brownish-yellow, in some the head is reddish-brown. Antennæ longer than body ; greenish-yellow to pale greenish-brown, some green at base, others at base of segment III., latter with thirteen to twenty-five sensoria, reaching nearly to apex, of varied sizes and arranged mainly in a line ; IV. a little shorter than III. ; IV. a little longer than V. ; VI. not quite so long as IV. plus V. Eyes deep red to black. Cornicles long, thin, pale green to yellowish-green, apices dusky, where there are a few reticulations and striæ, reaching beyond the cauda ; cauda pale yellowish-green, moderately long, four hairs each side and one dorso-apical one. Legs rather long, pale yellow-green, apices of femora and tibiae dusky ; tarsi dark. Wings with green to pale yellowish-brown insertions ; cubitus and stigma same colour.

Length, 2 to 2.5 mm.

Apterous Viviparous Female. Pale yellowish-green to various shades of green, some dull, others shiny. Antennæ very long, longer than body ; apices of segments dusky and a darkened area often around the group of sensoria on VI. ; apical segment usually dusky ; III. with one to five sensoria near base ; III. longer than IV. ; IV. a little longer than V. Cornicles pallid green to yellowish-green, cylindrical, long and thin, apices dusky, some faint apical reticulations.

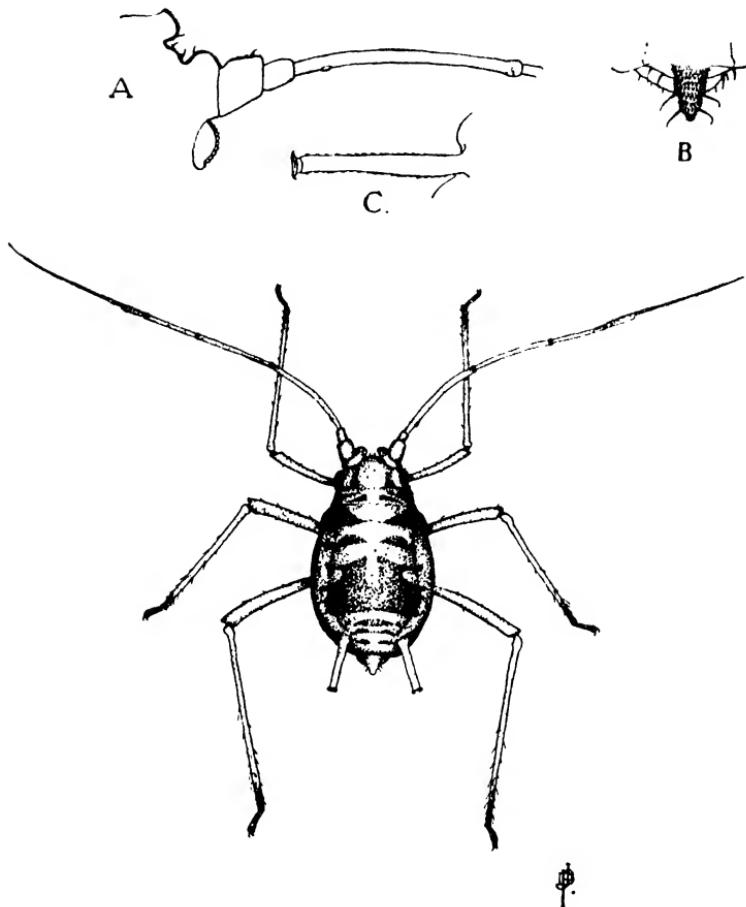


FIG. 99. *Myzus circumflexus* Buckt., apterous viviparous female. $\times 27$.

A. Head and antenna.

B. Cauda.

C. Cornicle. all $\times 60$.

Cauda pale yellowish-green, moderate size; four hairs on each side. Rostrum green, apex dusky, reaching to second coxae. Legs pale yellowish-green; apices of tibiae and tarsi dusky, a few short hairs on femora, many on tibiae; in some the apical banding is faint or almost absent (see Fig. 95, 4).

Length, 2.5 to 3 mm. (Theobald, 52).

Food Plants. *Aquilegia vulgaris* Walk., *Arum* sp., *Calceolaria*, *Chrysanthemum*, *Cineraria*, *Dianthus caryophyllus*, *D. barbatus*, *Geranium robertianum*, *G. molle*, *G. pusillum*, *Lilium* sp.,

Lycopersicum esculentum, *Malva rotundifolia*, *M. sylvestris*,
Primula veris, *Pelargonium*, *Persimmon*, *Pyrus germanica*, *Viola* sp.

Life History

Little is known of the habits of this aphid; in the British Isles it appears to be found only in glasshouses, where it is very common.

Viruses Transmitted by *Macrosiphum pelargonii*

Tulipa Virus 1, causing "break" of tulips.

Possibly the vector of **Pelargonium Virus 1**, causing leaf-curl of Pelargoniums.

Geographical Distribution. Widely distributed in Great Britain; it also occurs in the U.S.A.

Aphididæ (*continued*)

Myzus circumflexus Buckton. (*Myzus vincae* Gillette). Lily aphid.

DESCRIPTION

Alate Viviparous Female. Green; head and thorax dark; abdomen with various irregular dark markings, a common type being a semi-circular patch behind the scutellum, then five broken bands and two more prominent ones near the apex; in others the bands fuse before the cornicles to form a dark patch; on others the bands are more uniform, some are almost entirely green on the abdomen. Cauda black. Antennæ black, variable in length, as long as or slightly longer than body; segment III. with a line of about sixteen sensoria on one side, reaching to apex of segment, last two separated by a small space from others; IV. nearly as long as III., with three sensoria; V. shorter than VI.; VI. very thin, much longer than IV. plus V. Cornicles long and thin, black to dusky green, cylindrical, faint imbrication. Cauda green, spinose. Legs same colour as body; apices of femora, tibiae and the tarsi dark.

Length, 1·6 to 1·7 mm.

Apterous Viviparous Female (see Fig. 99). Bright, shiny, yellowish-green; thorax with one to two dark spots on each side. Abdomen with dark patches, one forming an irregular horseshoe-shaped area and a large black patch. In some, these typical markings are quite absent. Antennæ yellow to green, apices of segments III. to V. and all VI. dusky; frontal tubercles markedly porrected; antennal segment III. longer than IV., with a sensorium near base; V. a little shorter than IV., VI. longer than IV. plus V. Cornicles rather long and thin, yellow, in some dusky at apices; imbricated. Some colonies show no markings, except here and there, the insects being uniformly pale yellow or pale yellowish-green. In others the dark markings are very pronounced.

Length, 1·6 to 1·8 mm. (Theobald, 52).

Food Plants. Arum lilies, chrysanthemums, tulips, cyclamen, freesias, spiræas, cinerarias, schizanthus, persimmon, *Vinca major*,

Hordeum, Avena, Linum; most plants under glass are attacked; out of doors the Larger Periwinkle is the most attacked. Laing records it on *Oxalis*, *Alisma*, dahlia, watercress, *Lycium*.

Life History

This is one of the commonest greenhouse aphides in the British Isles, but the sexual forms do not appear to be known. It occurs every month of the year under glass, but increases most rapidly from February to May on arum lilies. Although mainly a glasshouse species, it will breed quite readily out of doors in midsummer.

Viruses Transmitted by *Myzus circumflexus*

Brassica Virus 3, causing mosaic of cauliflowers.

Cucumis Virus 1, causing mosaic of cucumber.

Nicotiana Virus 1, causing mosaic of tobacco (from tomato) only.

Solanum Virus 14, causing leaf-roll of potato.

Geographical Distribution. Widely distributed over Great Britain and Europe generally; North America and Buenos Aires.

Aphididæ (*continued*)

Myzus persicæ Sulz. The peach aphid, the spinach aphid, the green potato aphid.

DESCRIPTION

The following descriptions of the various forms of *M. persicæ* are taken from Theobald's *British Aphides*.

Adult Stem-mother. Pale green to almost pink, sometimes green with pink mottling, at others all green or all reddish. Antennæ of five segments, very pale, becoming dusky at apices, shorter than body, arising from prominent lobes; segment I. larger than II.; III. much longer than IV.; IV. a little shorter than V., which has the basal area about as long as the flagellum. Cornicles cylindrical, slender. Cauda green, moderately long, two pairs of lateral hairs and a sub-apical dorsal one. Legs pale, apices of tibiae and the tarsi dusky. Rostrum pale, apex dusky, reaching just past second coxae.

Length, 1·6 to 1·8 mm.

Apterous Viviparous Female. Various shades of green, yellow to pale brownish-pink; shiny, rather acuminate posteriorly, but ovate in general form. Antennæ slightly shorter than body, with well-marked frontal lobes on head; segment I. longer and wider than II.; III. about the same length as IV.; IV. longer than V.; VI. shorter than IV. plus V. Body often mottled because of young showing within. Cornicles green, yellow or pale pink, apices usually dusky; very slightly swollen either in middle or just below or above; rather long and thin, a few striæ across apices. Cauda pale, about one-third length

of cornicles, yellow or pinkish, three pairs of lateral hairs. Legs same colour as body ; apices of tibiae and the tarsi dusky, in some only the apices of tibiae. Rostrum reaches to or just past second coxae, apex dusky. A small lateral tubercle between cauda and cornicles. Subject to much variation in colour (see Fig. 98).

Length, 2 to 2·5 mm.

Alate Viviparous Female (Autumn migrant). Head, pronotal band and thoracic lobes black. Abdomen rather shiny, reddish to greenish-yellow, with a large almost black dorsal patch before cornicles and often passing between them ; one or two black bars in front and one or two behind it ; in some a single black bar in front with two spots cephalad ; four lateral black spots, one pair caudad of cornicles ; sometimes there are two pairs of large spots instead of bars in front. Antennæ about as long as body, black to brown, base of segment III. paler ; I. a little longer and much wider than II. ; III. a little longer than IV., with a line of eight to fourteen sensoria ; IV. much longer than V. ; VI. about as long as IV. plus V. Cornicles black to pale brown, slightly swollen towards middle or apical half, rather long, one to two terminal striae. Legs ochreous to reddish-green ; femora all or partly blackish ; also apices of tibiae ; tarsi black. Wings long, often as long again as body ; insertions and cubitus yellow to yellowish-green ; stigma grey to pale brown. Cauda dark, about two-thirds length of cornicles, with three pairs of lateral hairs and one dorso-apical ; two small wart-like processes behind cauda. Eyes black.

Length, 1·8 to 2·5 mm.

Alate Viviparous Female (Spring migrant). Very similar to the autumn alate female, but the cornicles are now and then slightly more cylindrical and black, as is also the cauda.

Oviparous Female (Apterous). Salmon-pink to pale brick-dust red ; apices of antennæ, tibiae and cornicles dusky. Tarsi black. Cornicles slightly swollen distally. Antennæ about two-thirds length of body. Hind tibiae broadened, with twenty to thirty sensoria.

Length 1·7 mm.

Male. Very similar to alate autumn female, but smaller and darker. Cornicles and cauda black, former slightly swollen. Antennæ longer than body ; III. longer than IV. ; IV. a little longer than V. ; VI. shorter than IV. plus V. ; sensoria along whole length of III., IV. and V.

Frontal lobes common.

Length, 1·8 to 1·9 mm.

Food Plants. It is not possible to give a complete list of the host plants of this aphid, since it is practically omnivorous. In Europe some of the important food plants are : *Brassica oleracea*, *Brassica* spp., *Beta vulgaris*, *Solanum tuberosum*, *Nicotiana* spp. and many other solanaceous plants, peach, nectarine, plum, cherry, almond, *Euonymus* sp., and tulips.

Life History

In England the eggs are laid in the axils of the buds of the peach, nectarine, *Daphne*, *Brassica* spp., and probably other

plants in October and November. The eggs are at first green, but later turn shiny black. They hatch out often as early as January on *Daphne* and on peaches under glass. The young are dark green, gradually becoming lighter, and by February the stem-mothers may occur, but usually not until March. By the time the blossom is out they commence to produce living young which are pale green and remain so, with now and then median and lateral darker lines. In May and June these become alate and fly off to cabbages and various other vegetables and flowers. In October they may produce an alate return migrant brood and these fly back to the peach, nectarine, cherry, *Daphne* and other woody plants. *M. persicæ* also hibernates as apteræ in considerable quantity on Brassicæ, particularly savoys. These plants constitute the main source of alate spring migrants in most rural districts. In heated glasshouses the aphides continue to breed asexually throughout the winter.

In the south and south-west of the United States of America and in South Africa, *M. persicæ* partly loses its migratory habit and feeds all the year round upon low-growing vegetables and weeds. In the north of the United States it normally hibernates in the egg stage upon various stone fruits. The life of an individual female aphis averages about thirty-six days, the approximate number of young produced being about forty.

Ecology. The effect of environmental factors upon the movements of *M. persicæ* in the field has been considered mainly in connection with the potato crop, because of the economic importance of the potato viruses transmitted by this aphis. At a temperature above 55° F.—approximately a minimum temperature during the day time of June and July in the British Isles—a relative humidity of 70 per cent and above will markedly reduce the instances of flight by *M. persicæ*. At higher temperatures of 80° F. and 90° F. the effect of humidity is even more marked, and flight is negligible when the humidity exceeds 85 per cent. Thus it follows that the districts in which low infestations of this aphis have been consistently recorded are low-lying, often almost at sea-level. Where data have been available for the centres, it has been found that high relative humidities are prevalent, and this would account for the low initial infestation of aphides on the potatoes. High altitudes with bleak exposed conditions are not necessarily the conditions in which aphides are scarce. Some recent laboratory experiments (11) have demonstrated the reluctance of winged *M. persicæ* to take

to the wing when the velocity of the wind exceeds three to four miles per hour. In other words, it is only when the wind velocity drops below this figure that the aphid expands its wings, rendering it buoyant, and prepares for flight. Only in such light breezes, therefore, can *voluntary migration* take place. So far as the problem of dissemination of virus diseases is concerned, it will be the prevalence of opportunities for this voluntary migration, when aphides can alight readily, take to the wing with ease and pass from plant to plant, that will be primarily involved.

Viruses Transmitted by *Myzus persicæ* Sulz.

Beta Virus 2, causing mosaic of sugar beet.

Brassica Virus 1, causing "ringspot" disease in cabbage.

Brassica Virus 2, causing mosaic in cruciferous plants.

Brassica Virus 3, causing mosaic in cauliflowers.

Matthiola Virus 1, causing mosaic in stocks.

Cucumis Virus 1, causing mosaic in cucumber and other plants.

Phaseolus Virus 1, causing "common mosaic" in beans.

Pisum Virus 2, causing "common mosaic" in peas.

Dahlia Virus 1, causing mosaic in dahlia.

Lactuca Virus 1, causing mosaic in lettuce.

Solanum Virus 2, causing leaf-drop in potatoes.

Solanum Virus 3, causing leaf-drop or mosaic mottle in potatoes.

Solanum Virus 8 (only in association with *Solanum Virus 3*), causing tuber-blotch in potatoes.

(Probably **Solanum Virus 9**, causing aucuba mosaic in potatoes.)

Solanum Virus 12, causing "spindle-tuber" disease in potatoes.

Solanum Virus 13, causing unniottled curly dwarf in potatoes.

Solanum Virus 14, causing leaf-roll in potatoes.

Solanum Virus 16, causing yellow dwarf in potatoes.

(Probably **Datura Virus 1**, causing a necrotic disease in *Datura Stramonium*).

Hyoscyamus Virus 1, causing mosaic in henbane.

Tulipa Virus 1, causing "breaking" in tulips.

Allium Virus 1, causing yellow dwarf disease in onions.

Iris Virus 1, causing stripe or mosaic in iris.

Geographical Distribution. *Myzus persicæ* is a cosmopolitan insect and is probably world-wide in its distribution. It is common all over Europe, North and South America, North and South Africa, Australia, New Zealand, Japan, India, Iraq and Bermuda.

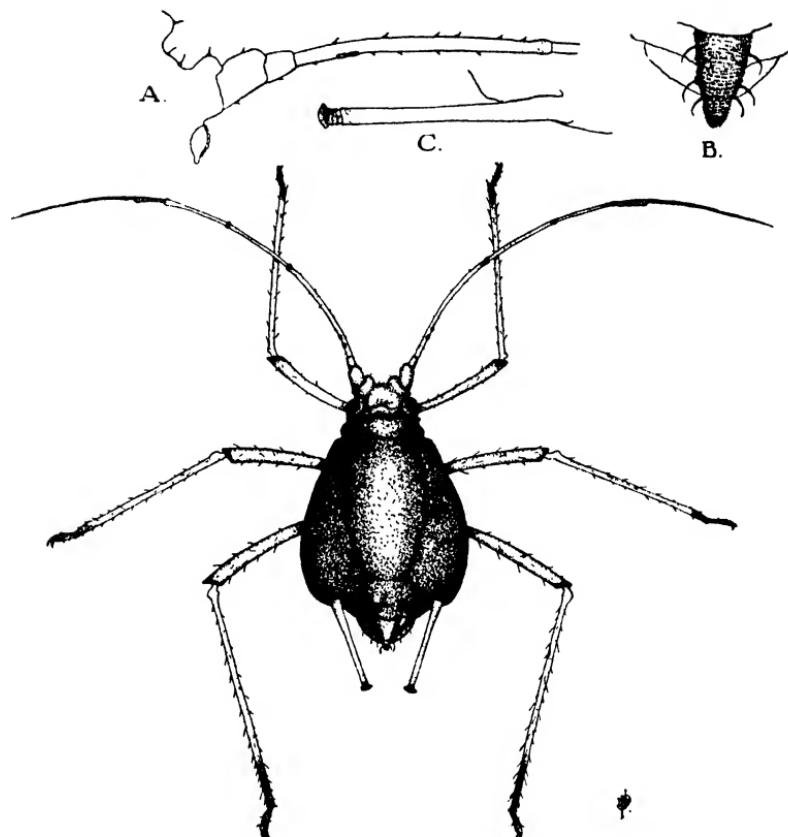


FIG. 100. *Myzus pseudosolani* Theob., apterous viviparous female. $\times 20$.

A. Head and antenna.

B. Cauda.

C. Cornicle. all $\times 45$. Note cylindrical cornicles.

Aphididae (continued)

Myzus pseudosolani Theob. (*Macrosiphum solani* Theob.).
Foxglove aphid, U.S.A.

DESCRIPTION

This aphid is very similar to *M. persicae*, but it can be differentiated by its pale cylindrical cornicles, and in the apterous female by the presence of one or two sensoria at the base of the third antennal segment. The frontal tubercles are less pronounced than in *M. persicae*, and the apices of the cornicles more widely flared and darker at the tips. It differs from *Macrosiphum gei* in the possession of non-reticulate cornicles (see Fig. 100).

Alate Viviparous Female. Green; head and thorax darkened; a pale band between head and pronotum and another between the pro- and meso-notum; thoracic lobes dark and a dark spot at base of each forewing. Abdomen with dark transverse bars, varying in form, and three dark lateral spots and another at base of cornicles; posterior parts of abdomen dusky green. Antennæ as long as, or a little longer, than body, darkened; segment III. with fourteen to twenty-three sensoria in a line. Cornicles green, apices dusky, with two to three apical striae, cylindrical. Cauda green. Legs green, base and apices of femora and apices of tibiæ and the tarsi darkened. Wings large, insertions yellow to green.

Length, 2·3 to 2·5 mm.

Apterous Viviparous Female. Green, yellow-green to deep green, in many rusty-red patches behind. Antennæ green, apices of segments III. to V. dusky; VI. dark; III. with one to two sensoria near base. Cornicles cylindrical, green, apices dusky; three striae near apex. Hairs of head and basal segment of antennæ capitate. Rostrum reaches second coxæ. Cauda about one-third length of cornicles (see Fig. 100).

Length, 1·8 to 2·5 mm. (Theobald, 52).

Food Plants. *Solanum tuberosum*, potato, *Digitalis purpurea*, foxglove.

Life History

The overwintering eggs are deposited on the garden foxglove (*Digitalis purpurea*). These hatch in spring and the resulting stem-mothers shelter in the folded leaves; later, winged females are produced, and these migrate to potato and other plants from June onwards, returning to the foxglove in September and October. *M. pseudosolani* is especially abundant upon sprouting potato tubers in company with *M. persicae*. It occurs from January to April upon the "seed" potatoes and on the haulm from July to October (36). The saliva of this aphis is toxic to some plants and produces much curling of the leaves and twisting of the petioles.

Viruses Transmitted by *Myzus pseudosolani*

Cucumis Virus 1, causing mosaic in cucumbers and other plants.

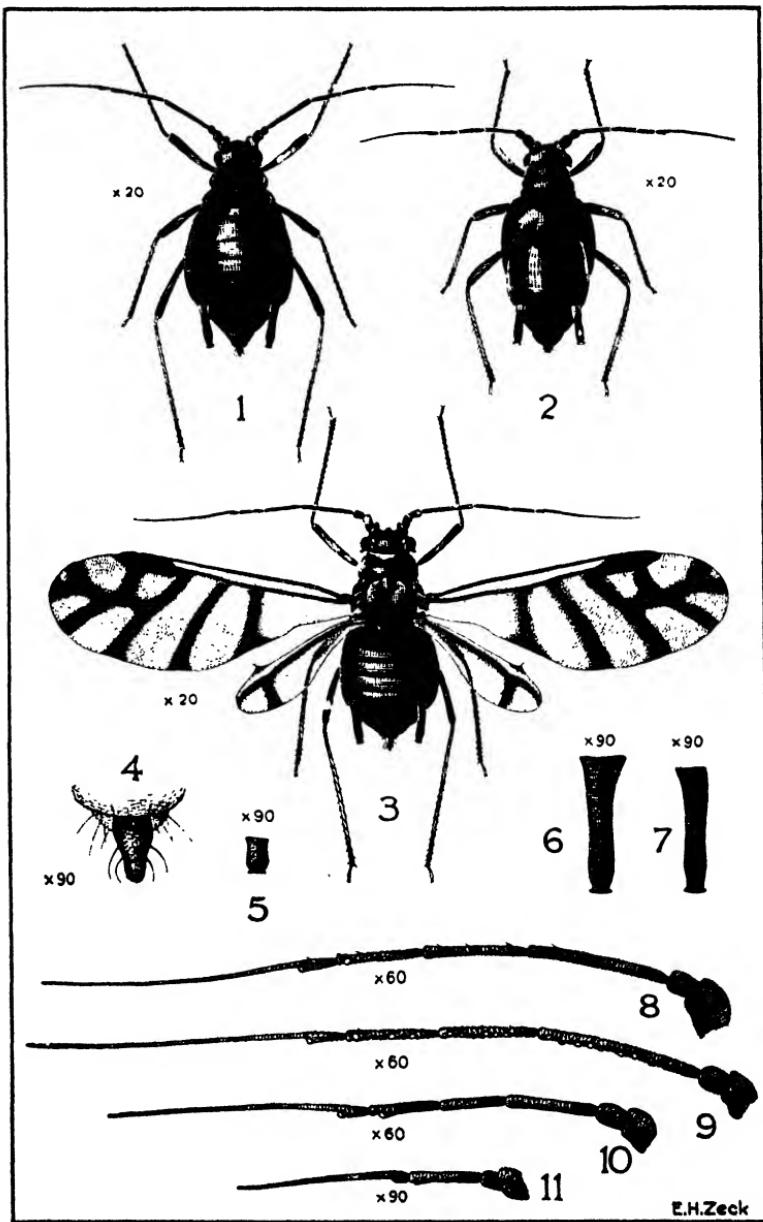
Nicotiana Virus 1, causing mosaic of tobacco (from tomato only).

Solanum Virus 14, causing leaf-roll in potatoes.

Geographical Distribution. Fairly widely distributed in the British Isles and in parts of the United States of America.

Aphididæ (*continued*)

Pentalonia nigronervosa Coq. The dark banana aphis.



DESCRIPTION

Sexual males and females and egg-laying forms of this species are not known. Adult viviparous females are both winged and wingless and they vary in colour from reddish to dark brown; the immature stages are of a lighter colour. The forewings have a peculiar venation unlike that of other members of the Aphididae, whilst the veins possess dusky to almost black borders easily visible with a hand lens (see Fig. 101). The hindwings are very small.

TECHNICAL DESCRIPTION

Alate Viviparous Female. Length of body, 1·2 to 1·6 mm.; wing expanse, 5 mm.; antennæ, 1·8 mm.; sensoria variable in number, III. with five to twelve (average eight), mostly on distal two-thirds; IV. with four to seven (average six), spaced along length; V. with one to three (average two), usual distal, one; small cluster at base of spur on VI. The wing venation is peculiar in that the radial sector has extended downwards and fused with the upper branch of the media, and has then turned again towards its natural course near the tip of the wing (see Fig. 101, 3).

Apterous Viviparous Female. Length of body, 1·2 to 1·6 mm.; antennæ, 1·7 mm.; V. with distal sensorium; VI. with cluster at base of spur (see Fig. 101, 1).

Nymph. Length of body, 1·5 mm.; antennæ, 1·4 mm.; V. with distal sensorium; VI. with cluster at base of spur (see Fig. 101, 2).

Larva (first stage). Length of body, 0·45 mm.; antennæ, 0·45 mm.; four-segmented; III. with one sensorium at distal end; IV. with one at base of spur (58).

Food Plants. *Musa sapientium*, *Musa banksii*, *Musa cavendishii* and other species of banana, *Musa textilis* (manila hemp), *Alpinia rafflesiana*, *Alpinia speciosa*, *Arum maculatum*, *Strelitzia* spp. and *Ravenala* sp.

Life History and Habits

The aphides are to be found clustered at the base of the pseudostem of the banana at soil level and for several inches

FIG. 101. *Pentalonia nigronervosa* Coq. The dark banana aphis. (Magnifications in Fig. to be reduced by half).

1. Apterous viviparous female.
2. Nymph of developing alate form.
3. Alate viviparous female.
4. Cauda of apterous viviparous female.
5. Cornicle of larva, first instar.
6. Cornicle of alate viviparous female.
7. Cornicle of alate viviparous female.
8. Left antenna of apterous viviparous female.
9. Left antenna of alate viviparous female.
10. Left antenna of nymphal form.
11. Left antenna of larva, first instar. (After Zeck.)

Agric. Gaz. N.S.W.)

below the surface, and also on young suckers just emerging from the ground. Dense colonies occur between the sheath of the outer leaf and the pseudostem, and also along the under surface of the leaves about the mid-ribs. Although the aphides flourish throughout the year, the summer, so far as New South Wales is concerned, is the period of greatest abundance.

There is no evidence that this aphis attacks the roots of the banana.

Ecology. Topography seems to exert an influence on the prevalence of the bunchy top disease of bananas and presumably of the aphis vector. In a plantation on a steep hillside in the early stages of the development of the disease it is noticed that bunchy top is usually more plentiful in the lower more sheltered portions of the plantation than on the exposed brow of the hill. The soil in the lower portion is usually of higher moisture content than the higher ground, the temperature is usually greater, and the growth of plants more vigorous. Aphides are usually most plentiful in such regions, the conditions being more favourable to their development than in higher exposed situations. They are most numerous during wet seasons; their numbers decrease rapidly during periods of drought (58).

Viruses Transmitted by *Pentalonia nigronervosa*

Musa Virus 1, causing bunchy top disease of bananas.

Musa Virus 2, causing bunchy top disease of manila hemp.

(Possibly **Musa Virus 3**, causing mosaic disease of bananas.)

Geographical Distribution. *P. nigronervosa* was first described and figured from specimens infesting bananas growing at St. Denis (Isle Bourbon) by the French entomologist Coquerel in 1859. It has since been recorded from many parts of the world as follows: United States of America (Wilson, 1909), Australia (Froggatt, 1923; Magee, 1927; Hardy, 1928), Bermuda (Ogilvie, 1925), Brazil (Moreira, 1925), England, Kew (Laing, 1922), Formosa (Takahashi, 1928), Hawaii (Fullaway, 1910; Zeck, 1926), Fiji (Magee, 1926), India (Theobald, 1923; George, 1928), Jamaica (Gowdey, 1925), Philippine Islands (Ocfemia, 1927), Trinidad, Egypt (Laing).

Aphididæ (continued)

Rhopalosiphum (Aphis) pseudobrassicæ Davis. The false mealy or cabbage aphis.

DESCRIPTION

Apterous Viviparous Female (Fig. 91, 4). Entire body pale whitish green, head slightly dusky. Abdomen with a longitudinal row of impressed dots along each side in line with the cornicles; also on each side of the median dorsal line is a row of transverse shining areas with a reticulated surface, those on the last four or five segments usually united; and a similar row of smaller areas on each side. These shining reticulated areas contrast with the rest of the body which is dull and very slightly pulverulent. Thoracic segments with similar transverse areas. In specimens just moulted the entire body appears shining and reticulated.

Eyes black. Antennæ blackish excepting segments I., II. and basal half of III., which are pale; reaching a little beyond the middle of the body; segment III. longest, being one-half to three-fourths longer than filament VI.; segments V. and VI., base with the usual distal sensoria. Beak reaching to coxae of second pair of legs. Legs pale with dusky joints, the tips of the tibiae and all of the tarsi black. Cornicles pale with the tip dusky, slightly swollen towards the tip and constricted just before the apex, and noticeably longer than the cornicles of *Brevicoryne brassicae*. Cauda conical, and dusky to blackish. Length of body, 1.66 mm.; antenna, total average length, 1.311 mm. (13).

Alate Viviparous Female (Fig. 91, 3). Head and thorax black. Abdomen pale apple green with a tint of nile green and a row of three black spots on each side anterior to the cornicles; a row of small impressed dots on each side dorsad of the larger spots; and in addition a few scattered inconspicuous dusky markings on the dorsum, and the last three segments with black transverse, dorsal median markings.

Eyes black. Antennæ black; almost reaching to base of cornicles; segments III. and VI., filament subequal; segment III. with nineteen to twenty-six moderately tuberculate circular sensoria irregularly placed; IV. with six to ten, often more or less in a row; V. and VI.. base with the usual distal sensoria and not infrequently segment V. bears one or two near the base. Wings with black and rather conspicuous veins, and the terminal branch of the media nearer the apex of wing than where it first branches. Legs with femur pale brownish to blackish, tibia pale brownish with tip black and tarsus black. Cornicles dusky, paler at tips, and shaped as in the apterous form. Cauda concolorous with the abdomen or paler. Length of body, 1.4 mm.; length of wing, 2.4 mm.; antenna, total average length, 1.833 mm. (13). The winged female can be separated from *B. brassicae* by the sensoria on antennal segment IV. in the former.

Food Plants. Various cruciferous plants, both wild and cultivated, especially *Brassica* spp. In New Zealand it has only been observed on cruciferous weeds.

Life History

There is little information on the habits of *R. brassicae*; in the United States it is usually most plentiful on cruciferous crops in

the autumn, and Davis suggests that the winter is probably passed as viviparous females.

Viruses Transmitted by *Rhopalosiphum pseudobrassicæ*

Brassica Virus 3, causing mosaic of Brassicæ.

Matthiola Virus 1, causing mosaic in stocks.

Phaseolus Virus 1, causing common mosaic in beans.

Allium Virus 1, causing yellow dwarf in onions.

Geographical Distribution. This aphis seems widely distributed in North America ; it has also been recorded from British Columbia, Nova Scotia, Ontario, New Zealand, New South Wales and Japan. There do not seem to be any records of its occurrence in Europe.

ARACHNIDA Eriophyidæ

Eriophyes ribis (Westw.) Nal. The black currant gall mite.

DESCRIPTION

Body much drawn out, cylindrical. Shield 34μ long, triangular, anterior edge projecting forwards and partly covering rostrum, lateral edges very slightly projecting. Thoracic shield marked with five longitudinal furrows which are drawn through central portion of shield. Sides of shield strongly punctate and marked with indistinct longitudinal curved lines. Thoracic bristles absent. Rostrum short, 16μ long, stout, sloping towards apex. Chelicera 20μ long. First pair of legs 36μ long, fourth and fifth joints almost equal in length, 16μ long ; tarsal bristles 18μ , femoral bristles 24μ , claws 8μ long. Second pair of legs 33μ ; fourth and fifth joints 14μ ; bristles strong ; feathered claws five-rayed, 6μ long, delicate ; claws 10μ long.

Sternal ledge deeply forked. First pair of coxal bristles situated behind anterior edge of sternal ledge, short ; second pair placed before the inner coxal angle ; the third, 40μ long, moderately stout.

Abdomen marked with punctate rings on dorsal and ventral surfaces, there being seventy rings. Lateral bristles 30μ long, placed in line with genital organ. First pair of ventral bristles 45μ long, second pair 9μ , third pair 15μ , fine, hair-like. Anal lobe moderately small, conspicuous. Caudal bristles prominent, long, tapering hair-like towards apex. Accessory bristles absent. Epigynum raised, basin-like, 21μ broad. Coverflap marked with longitudinal lines. Genital bristles situated at lateral edges, short.

Female, 220μ long, 40μ broad ; male, 150μ long, 38μ broad (32).

Food Plants. *Ribes nigrum* L. ; *R. rubrum* L. ; *R. alpinum* L. ; *R. grossularia* L. ; *R. sanguineum* Pursh.

Life History and Habits

The following description of the life history of this mite is taken from the work of Massee (32). The immature and adult mites enter the newly forming buds towards the end of May, the first date noted being May 25th. As a rule only two or three mites enter one bud. They pierce their way to the centre of the bud and immediately commence to breed, eggs being found within three or four days of the entry of the mites. In an average season the swollen or "big" buds may be seen on the bushes by the third week in August. It is not, however, always possible to determine which buds are inhabited by mites before the end of July, and even then it may be necessary to dissect the buds and examine them microscopically. The eggs of the mite are present all the year round. Immediately after the migration period, eggs are deposited on the shoots, and during the flowering season they are frequently laid in the blossoms. They are laid on the leaves and shoots during the summer months. The duration of the egg stage varies very considerably, according to the season. The eggs hatch within three to seven days during the spring and summer months; during the winter the egg stage is extended over a period of three to four weeks. The ova are very large in comparison with the adult mite. Just before an egg hatches it tends to become drawn out, and the form of the mite is visible through the transparent chorion.

The spring migration period depends upon the weather conditions prevailing at the time. The earliest record is April 1st, but migration may be delayed until the middle of May; as a general rule it reaches its height during the latter part of April or in May. No autumn migration has been observed in England. The mites are disseminated from bush to bush partly by the wind and partly through the agency of insects. This process is aided by the peculiar leaping habits of the mites.

E. ribis responds very readily to temperature, especially under artificial conditions, but cold weather and frosts do not appear to affect the mites to any great extent.

Virus Transmitted by *Eriophyes ribis*

Ribes Virus 1, causing reversion of black currant bushes.

Geographical Distribution. British Isles, Central Europe, British Columbia; abundant and widely distributed.

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CHAPTER IX

SUSPECTED VIRUS DISEASES REQUIRING FURTHER INVESTIGATION

IN this chapter brief notes are given of a number of apparent virus diseases which still require investigation.

Ranunculaceæ

Clematis vitalba. On several occasions plants of clematis have been observed in England showing symptoms of a mosaic-like disease. The leaves exhibit a yellowish mottle which sometimes takes the form of chlorotic rings. The general appearance of the disease is somewhat similar to the disease caused in peonies by *Paeonia Virus 1* (see p. 5).

Cruciferæ

Lunaria sp. "Honesty." A mosaic disease of honesty has been recorded by Ogilvie (*in litt.*), and the writer has also received from Scotland specimens of similarly affected plants. The leaves show a diffuse light and dark green mottle and the flowers are frequently "broken." The virus causing this disease does not appear to be transmissible to *Nicotiana* spp.

Violaceæ

Viola spp. Violets, both wild and cultivated, are susceptible to a mosaic disease which produces a mottle on the leaves and a "break" in the flower colour in the form of white flecks.

Polygonaceæ

Rheum officinale L. Rhubarb. Rhubarb in England and elsewhere is sometimes affected with a mosaic-like disease in which pale chlorotic areas develop on the leaves, together with much stunting of the plant.

Oleaceæ

Syringa vulgaris. Lilac. (1) *Ringspot*. The lilac seems to be susceptible to more than one virus. Atanasoff (2) describes a

ringspot disease affecting this plant in North Bulgaria. The leaves of affected plants show numerous rings of a lighter green colour. The rings may be small, and when very numerous unite to give the leaves a marbled appearance. Usually the rings are discrete and are scattered uniformly over the leaf blade, being visible from both sides of the leaf. Where the rings are very numerous, along the leaf margin, a general chlorosis develops, followed by the death of the leaves. There is no rolling or leaf distortion.

(2) *Lilac leaf-roll*. The writer has received from Mrs. Alcock, in Scotland, some lilac plants which exhibited symptoms suggestive of virus infection. The leaves showed a pallor and extreme stiffness; they were very brittle and "tinny," and there was a certain amount of rolling. Owing to their brittle nature, much breakage of the leaves takes place, with the result that in a long-infected plant the leaves tend to hang down in strips. There appears to be considerable starch accumulation in the leaves.

In the young foliage developing the following season, rolling appears very shortly after the unfolding of the leaves. Frequently only one side of the leaf rolls; as a rule the rolling is in a downward direction, though upward rolling has also been observed. The flowers are poor in quality and may fail to develop. The writer was informed by the grower, in whose nursery the diseased plants were found, that the infection had spread among the young lilac plants in the field.

It is not certain whether this is the same disease as the "mosaic" of lilac recorded by Atanasoff (2). The rolling of the leaves in the two diseases seems similar, but the writer has not observed the mosaic mottling described by Atanasoff.

Jasminum officinale. Jasmine. An infectious variegation of jasmine has been known since the latter part of the seventeenth century. This variegation has been transmitted by grafting from *Jasminum revolutum* to *J. officinale* and *vice versa* (2).

Malvaceæ

Malva sylvestris L. Common mallow. The writer has received specimens of the common mallow affected with an apparent mosaic disease. The leaves showed patches of bright yellow interspersed with dark green, they were slightly distorted and occasionally showed blistering.

Caryophyllaceæ

Dianthus caryophyllus. The carnation. This plant is subject

to an apparent virus disease of the mosaic type in which the leaves are mottled and the flowers are "broken." It has been recorded in England by Ainsworth and Ogilvie.

Rosaceæ

Pyrus malus L. Apple. The *bitter pit* disease of apples is considered by Atanasoff (1) to be due to a virus, since the leaf mottling which accompanies bitter pit is transmissible by grafting and is thought to be one of the symptoms of the disease. Atanasoff suggests that a virus similar to that causing mosaic of plums (see p. 146) is concerned.

Cannabinaceæ

Humulus lupulus L. The hop. Salmon has described (12) one or two obscure diseases of the hop which may be due to virus infection. "Fluffy-tip" is a condition in which the stipules are prominent and the leaves small at the tips of the bines, giving a feathery appearance. The leaves are also of a pale yellow-green colour. In "split leaf mottle" the leaves show pale areas where splits develop, prominent yellow blotches are also present. In the disease known as "small hop," which appears sporadically on single plants, the lateral branches are usually devoid of leaves and the cones remain small and seldom exceed $\frac{1}{2}$ inch in length; masses of densely crowded buds may occur on the root stock at the base of the bine.

Juglandaceæ

Hicoria pecan. Native pecan. *H. aquatica*. Water hickory.

The so-called "bunch disease" of pecan is thought to be caused by virus infection. The chief symptoms of the disease are the brooming of branches and shoots, early foliation of diseased branches in the spring, chlorotic, thin, broad, wavy and flexible leaves, and, in later stages, dying back of the branches. The disease has been transmitted by grafting.

The Schley and Mahan varieties are very susceptible, while the Stuart variety is highly resistant.

The bunch disease is known to be present in Louisiana, Mississippi, Oklahoma, and Texas, U.S.A. (6).

Leguminosæ

Laburnum vulgare. Laburnum. The laburnum is sometimes affected with an infectious variegation. The leaves are mottled

with lemon-yellow spots and streaks; the spots are small and scattered between the veins. The streaks are very prominent and always run along the main lateral veins. Gradually some of the leaves take on a bright yellow colour (2).

Caprifoliaceæ

Sambucus nigra. Elder. Blattný (5) has described a mosaic disease of the elder. The leaves of infected plants show a dark yellowish-green veining. The plants are dwarfed with few, mostly sterile, flowers. The insect vector is said to be *Aphis sambuci*.

Rutaceæ

In a recent publication Atanasoff (3) discusses a number of diseases affecting the *citrus*, and offers some facts suggesting that these diseases are of virus origin. The orange is subject to an infectious chlorosis characterised by clearing of the leaf-veins and rapid decline and death of the infected tree. It is thought that this chlorosis may be the same as the Mediterranean disease known as "mal secco" and the Florida citrus disease called "blight" or "wither tip."

Another disease of the orange considered by Atanasoff to be due to virus infection is the so-called "little-leaf" disease. He describes the symptoms as follows. The lower leaves of the annual shoots are darker green, much smaller, severely curled and leathery, while the top leaves usually are more or less normal, but may be longer than healthy leaves. All affected leaves show a very plain marbling on the upper side. On the lower side of the leaves are seen greenish-white streaks and blotches. On the upper surface of the leaves the marbling consists of a greater variation of colours, ranging from dark green to light green or almost white. The affected leaves are usually very small, deformed and curled, they are also longer than normal. Petri has suggested that this disease is spread by the aphid *Toxoptera aurantis* Boyer which attacks the citrus buds. Other diseases of citrus which are considered to be due to virus infection are "crinkly leaf," "spot mosaic," "zonate chlorosis" and "ring blotch."

Umbelliferæ

Pastinaca sativa L. Parsnip. The writer has observed a mosaic disease on the common parsnip. Affected leaves show a mottle of light green and yellow or almost white patches.

Precisely similar symptoms have also been observed on certain umbelliferous weeds.

Compositæ

Taraxacum vulgare. Dandelion. In the Cambridge, England, district the dandelion is frequently found affected with a disease in which yellow patches develop on the leaves. These patches occur as isolated spots here and there on the leaf surface. They are not numerous enough as a rule to produce a mottle. This disease was first described in America by Morse (11), who called it dandelion " yellows."

Scrophulariaceæ

Digitalis purpurea. Foxglove. The foxglove is susceptible to a mosaic disease which produces a marked mottling of the leaves. Often the whole area of a leaf where two veins meet is completely yellow. Light green patches are also distributed uniformly over the other parts of the leaf, which is a little smaller than normal. The insect vector appears to be an aphis, but the species is as yet undetermined (J. Grainger, *in litt.*).

Solanaceæ

Cestrum parqui L'Hérit. Hedges of this plant growing in different parts of the Campagna in Italy have been observed to show leaf abnormalities consisting of various combinations of surface wrinkling, edge waving and chlorosis, apparently due to virus attack. The cells of the affected tissues show inclusions and other cytological modifications characteristic of a virus disease. It is suggested that a virus may have been acquired from potato crops in the vicinity (14).

Solanum tuberosum. Potato. A suspected virus disease of the potato known as " giant hill " is prevalent in certain parts of the United States of America. The disease is characterised in affected plants by abnormally large, coarse vines. This symptom generally does not become evident until the plant is mature. Affected plants remain green longer than healthy plants and also are somewhat resistant to frost. Tubers from diseased plants are generally large and often knobby and cracked. Further work is needed to determine whether " giant hill " is a virus disease or a genetic abnormality (15).

Lycopersicum esculentum. Tomato. A ringspot type of disease in tomatoes occurs in many parts of Indiana, U.S.A. The

disease is characterised by intricate patterns of brown necrotic rings and lines on young leaves, broad, sunken, necrotic streaks on petioles and stems of young shoots, necrosis of shoot terminals, and often corky-brown necrotic rings on green and ripe fruits.

Infected tomato plants seem to recover from the disease, but retain the virus in infectious form. High temperatures increase the severity of the disease. Mechanical transmission of the virus is best effected by using carborundum abrasive. The virus is transmissible to *Datura* from tomato by mechanical inoculation, but is only transmissible back to tomato by grafting or by first passing the virus through tobacco.

The thermal death-point of the virus lies between 56° and 58° C. for ten-minute exposures. The longevity *in vitro* at room temperature is between twenty-one and twenty-seven hours. Dilutions of 1 : 500 render the virus non-infectious. This virus has been transmitted to, and recovered from, fourteen species in the Solanaceæ and one species in the Amaranthaceæ (9). The relationship of this virus, if any, with tomato viruses already described is not known.

Bignoniaceæ

Tabebuia pallida. Witch's broom. This disease, which has previously been attributed to a fungus, has now been shown to be due to a virus which is transmissible by tissue inoculation. It is not seed-transmitted (7).

Amaryllidaceæ

Hippeastrum equestre Herb. A mosaic disease of Hippeastrum has been described from time to time, but there does not appear to be any information on the properties or affinities of the virus which causes the disease. An outstanding feature of this mosaic is the occurrence in the diseased tissue of large and characteristic intracellular inclusions, or X-bodies (8, 10).

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APPENDIX

IN the following appendix are given the most characteristic symptoms of the various virus diseases on the more important host plants. It is hoped that this appendix will enable the student to arrive at a preliminary diagnosis of a suspected virus disease and also help him, by means of the page references, to find in the book the information on all the viruses which attack any one particular plant. By this means, also, the disadvantage to the practical man of the classification of the viruses, rather than the diseases they produce, will be partly overcome.

Host Plant	Symptoms	Disease Caused by
<i>Adonis aestivalis.</i>	Plant dwarfed, chlorotic, with many secondary shoots. Leaves small, short.	Aster Yellows Virus (<i>Callistephus Virus 1</i>), p. 216.
<i>Allium cepa,</i> Onion.	Plant stunted, brittle, yellow. Young leaves show chlorotic streaks. Old leaves chlorotic with yellow markings.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 52.
" "	Leaves yellow, crinkled and somewhat flat, drooping flower stems, crinkled and streaked with yellow.	Onion Yellow Dwarf Virus (<i>Allium Virus 1</i>), p. 419.
<i>Althaea rosea,</i> Hollyhock.	Outgrowths (enations) on leaf veins, leaves thick and downwardly curled, plant with "bunchy-top" appearance.	Cotton Leaf-curl Virus (<i>Gossypium Virus 1</i>), p. 93.
<i>Amaranthus auroro.</i>	Plants stunted and chlorotic, with many short secondary shoots. Leaves show vein-clearing, and in <i>A. auroro</i> turn from red to yellow-grey.	Aster Yellows Virus (<i>Callistephus Virus 1</i>), p. 216.
<i>Amaranthus caudatus.</i>		
<i>Amaranthus retroflexus,</i> Pigweed.	Plant dwarfed (8 to 10 inches), usually with only one stem, and abnormally spreading habit. Leaves mottled yellow-green, small, narrow, irregular, petioles long, internodes short. Amount of seed reduced.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 57.
<i>Ananas cosmosus,</i> Pineapple.	A slightly raised circular yellowish spot on the upper surface of a young leaf. Below this spot a yellowish streak develops. Leaves stunted, chlorotic and brittle.	Pineapple Yellow or Spot Virus (<i>Ananas Virus 1</i>), p. 403.

Host Plant	Symptoms	Disease caused by
<i>Anemone nemorosa.</i>	Plant shows spreading habit, leaves thickened and misshapen, indentations shallow. Flowers suppressed or deformed.	Anemone Alloio-phylly Virus (<i>Anemone Virus 1</i>), p. 6.
<i>Antirrhinum majus,</i> Snapdragon.	Necrotic spots (or rings) on leaves, spots about 1 mm. in diameter with a light centre, surrounded by a band of dark brown tissue. Plant usually killed.	Tobacco Ringspot Virus (<i>Nicotiana Virus 12</i>), p. 279.
" "	Concentric rings or zoned spots on leaves, some necrosis, plant not killed.	Tomato Spotted Wilt Virus (<i>Lycopersicum Virus 3</i>), p. 314.
<i>Apium graveolens,</i> Celery.	Plant stunted with flattened, open appearance due to outward and downward curling of petioles. Leaflets show yellowing along veins, with dark green thickened areas, causing crinkling. Petioles often develop buff spots, and become brown and shrivelled.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 66.
" "	Young leaves with shortened, twisted, chlorotic petioles. Older leaves yellowed or blanched, petioles longer than normal, upright, but later becoming flattened out and brittle. In advanced stages, heart decays.	Celery Yellows Virus. (<i>Callistephus Virus 1A</i>), p. 222.
" "	Bold mosaic mottling on the leaves; leaflets with cleared veinlets.	Western Celery Mosaic Virus (<i>Apium Virus 1</i>), p. 204.
" "	Leaves with a striking chlorosis; green islands of tissue in lemon-yellow areas.	Celery Calico Virus (<i>Apium Virus 2</i>), p. 205.
<i>Aquilegia</i> sp. Columbine.	Plant stunted. Leaves show vein-clearing followed by mottle. Old leaves necrotic. Flowers few or absent.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 55.
<i>Arabis</i> sp., Rockeress.	Leaves mottled with yellow ring markings.	Cabbage Ringspot Virus (<i>Brassica Virus 1</i>), p. 10.
<i>Arachis hypogaea</i> .	Mosaic mottling of leaves, plant strikingly dwarfed, forming a	Rosette Disease Virus (<i>Arachis</i>

Host Plant	Symptoms	Disease caused by
Groundnut, Peanut.	close tuft of small leaves. Flowers sometimes present, but seed not formed.	<i>Virus 1</i>), p. 185.
<i>Asclepias syriaca</i> , Milkweed.	Plant stunted (2 feet or less). Leaves mottled with greenish-yellow patches, distorted, lanceolate, sometimes abnor-mally long and narrow, margins tend to curl up.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 67.
<i>Barbarea vulgaris</i> , Cress.	Leaves curled, petioles shortened, elevations on lower surfaces.	Sugar Beet Curly Top Virus (<i>Beta Virus 1</i>), p. 24.
<i>Begonia</i> sp.	Rings or zoned spots on leaves, some mottling, plants stunted, flowers poor.	Tomato Spotted Wilt Virus (<i>Lycopersicum Virus 3</i>), p. 299.
<i>Beta vulgaris</i> , Sugar Beet.	Leaves curled with blister-like elevations ; wart-like protuber- ances on lower surfaces ; vein- clearing in young leaves ; exudations from petioles, mid- ribs and veins ; yellowing.	Sugar Beet Curly Top Virus (<i>Beta Virus 1</i>), p. 25.
“ “	Plant stunted and deformed. Heart leaves develop yellow spots, later a bright mottle, with distortion of leaf surface. Leaf tips bent back, margins crinkled.	Sugar Beet Mosaic Virus (<i>Beta Virus 2</i>), p. 38.
“ “	Plant stunted. Leaf-veins swollen, crooked, translucent ; leaves crinkled. Old leaves die prematurely.	Sugar Beet Leaf- curl Virus (<i>Beta Virus 3</i>), p. 42.
“ “	Young leaves unaffected. Old leaves yellowed, brittle, short and thick, with accumulation of starch.	Sugar Beet Yellows Virus (<i>Beta Virus 4</i>), p. 45.
“ “	Leaves dwarfed, down-curled, savoyed ; veins cleared, thickened. Roots discoloured.	Sugar Beet Savoy Disease Virus (<i>Beta Virus 5</i>), p. 47.
“ “	Leaves with faint light-coloured zigzag lines and patterns. No necrosis.	Tobacco Ringspot Virus (<i>Nicotiana Virus 12</i>), p. 278.
<i>Bidens discoidea</i> , Spanish Needle.	Leaves with very fine zigzag lines, sometimes forming enclosures with irregular borders, tissue in the marginal lines becomes necrotic and turns white.	Tobacco Ringspot Virus (<i>Nicotiana Virus 12</i>), p. 275.

Host Plant	Symptoms	Disease caused by
<i>Brassica alba</i> , White Mustard.	Plants stunted. Leaves yellow, show streak symptoms.	Crucifer Mosaic Virus (<i>Brassica Virus 4</i>), p. 17.
<i>Brassica Chinensis</i> , L. Chinese Cabbage.	Plant stunted. Leaves mottled, distorted ; margins irregular. Flowers reduced.	Crucifer Mosaic Virus (<i>Brassica Virus 4</i>), p. 17.
<i>Brassica napobrassica</i> , Swede.	Plant stunted, leaves yellow mottled ; no streak symptoms.	Crucifer Mosaic Virus (<i>Brassica Virus 4</i>), p. 17.
<i>Brassica napus</i> , Rape.	Plant stunted, leaves mottled.	Crucifer Mosaic Virus (<i>Brassica Virus 4</i>), p. 17.
<i>Brassica nigra</i> , Black Mustard.	Plants stunted. Leaves yellow, show streak symptoms.	Crucifer Mosaic Virus (<i>Brassica Virus 4</i>), p. 17.
<i>Brassica oleracea</i> L., var. <i>capitata</i> L. Cabbage.	Mosaic mottle on older leaves, becoming black and necrotic in the ringspot appearance.	Cabbage Ringspot Virus (<i>Brassica Virus 1</i>), p. 9.
" "	Leaves with blotchy dark green mottle. No necrosis.	Turnip Mosaic Virus (<i>Brassica Virus 2</i>), p. 13.
<i>Brassica oleracea</i> L., var. <i>botrytis</i> L., Cauliflower and Broccoli	Diffuse mottling, with other symptoms as on cabbage.	Cabbage Ringspot Virus (<i>Brassica Virus 1</i>), p. 9.
" "	Leaves show vein-clearing, then vein-banding, with dark green patches between veins. Small necrotic lesions appear later.	Cauliflower Mosaic Virus (<i>Brassica Virus 3</i>), p. 15.
" "	Plants stunted. Leaves mottled or blotched with yellow.	Crucifer Mosaic Virus (<i>Brassica Virus 4</i>), p. 18.
<i>Brassica oleracea</i> L., var. <i>gem-</i> <i>mifera</i> , Zenker, Brussels Sprouts.	Plants stunted. Leaves mottled or blotched with yellow ; ring- spot effect appears later.	Crucifer Mosaic Virus (<i>Brassica Virus 4</i>), p. 18.
<i>Brassica rapa</i> , White Turnip.	Plant stunted. Leaves yellow mottled. No streak symptoms.	Crucifer Mosaic Virus (<i>Brassica Virus 4</i>), p. 17.
<i>Calceolaria</i> sp.	Plant dwarfed, chlorotic. Sec- ondary shoots remain small. Leaves much reduced in size.	Aster yellows Virus (<i>Callistephus Virus 1</i>), p. 221.

Host Plant	Symptoms	Disease caused by
<i>Calceolaria</i> sp.	Large, pale, irregular blotches on the leaves, leaf outline distorted, green tissue between the blotches sometimes raised in blisters; red or pinkish necrosis near the veins.	Tomato Spotted Wilt Virus (<i>Lycopersicum Virus 3</i>), p. 308.
<i>Calendula</i> sp.	Plant mottled, distorted; leaves with necrotic areas. Flowers small, misshapen.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 67.
„ „	Leaves show pale zigzag lines or rings.	Tobacco Ringspot Virus (<i>Nicotiana Virus 12</i>), p. 275.
<i>Callistephus Chinensis</i> . China Aster.	Leaves mottled or entirely yellow. Flower heads small, malformed, abnormally numerous.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 67.
„ „	Plant dwarfed with upright habit. Abnormal production of secondary shoots. Growth increased and suppressed in different parts of plant. Leaves with pronounced chlorosis (not mottle), vein-clearing, slight malformation, with lengthening of petioles. Flower heads dwarfed, petals often become green. Flowers may develop into vegetative shoots.	Aster Yellows Virus (<i>Callistephus Virus 1</i>), p. 217.
„ „	Central leaves distorted, dark green mottle on leaves and flower bracts; flowers small and distorted.	Tomato Spotted Wilt Virus (<i>Lycopersicum Virus 3</i>), p. 300.
<i>Campanula pyramidalis</i> .	Concentric rings or wavy lines and markings on the leaves, plants stunted.	Tomato Spotted Wilt Virus (<i>Lycopersicum Virus 3</i>), p. 304.
<i>Capsella bursa-pastoris</i> , Shepherd's Purse.	Plants stunted, seed stalks twisted, fruits malformed.	Curly Top Virus (<i>Beta Virus 1</i>), p. 24.
<i>Capsicum annuum</i> .	Plant compact, sometimes rosetted. Young leaves curled downward, mottled; older leaves narrow with filiform tips. Internodes and petioles short. Fruits sometimes with wart-like swellings.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 70.
„ „	Plant dwarfed, distorted. Leaves show local spotting and ring-	Tomato Ring Mosaic Streak

Host Plant	Symptoms	Disease caused by
<i>Capsicum frutescens</i> , Pepper.	like patterns of green and yellow.	Virus (<i>Lycopersicum Virus</i> 2), p. 295.
<i>Cheiranthus Allioni</i> .	Young leaves curled inwards; veins cleared, with minute swellings; older leaves cupped outward.	Sugar Beet Curly Top Virus (<i>Beta Virus</i> 1), p. 32.
<i>Cheiranthus cheiri</i> L., Wallflower.	Plant chlorotic, abnormally branched. Leaves longer and narrower than normal.	Aster Yellows Virus (<i>Callistephus Virus</i> 1), p. 216.
<i>Chenopodium album</i> , White Goosefoot.	Plant stunted. Leaves mottled and crinkled. Flower colour "broken."	Cabbage Ringspot Virus (<i>Brassica Virus</i> 1), p. 10.
<i>Chrysanthemum</i> spp.	Young leaves develop isolated yellow spots, with curling-under of margins. Affected leaves shrivel and die.	Sugar Beet Mosaic Virus (<i>Beta Virus</i> 2), p. 41.
<i>Cineraria hybrida</i> .	Young plants with pale chlorotic areas on the upper leaves, followed by bronzing; leaves and stem later show necrosis; older plants with bronzing or "rustiness" also present, together with slight mottling.	Tomato Spotted Wilt Virus (<i>Lycopersicum Virus</i> 3), p. 302.
" "	Plant stunted, chlorotic. Numerous upright secondary shoots. Leaves with vein-clearing; petioles elongated.	Aster Yellows Virus (<i>Callistephus Virus</i> 1), p. 220.
<i>Citrullus vulgaris</i> , Watermelon.	Pale yellow spots on the leaves, followed by browning of the veins and frequently death of the plant.	Tomato Spotted Wilt Virus (<i>Lycopersicum Virus</i> 3), p. 302.
" "	Plant stunted, yellow; terminal leaves dwarfed, puckered, curled outward, deep green; older leaves yellow.	Sugar Beet Curly Top Virus (<i>Beta Virus</i> 1), p. 29.
" "	Plant shows proliferation of shoots round the crown, with shortened internodes and crowding of young leaves. Leaves with diffuse mottle and sometimes extreme malformation and reduction of lamina. Old leaves with conspicuous mottle. Flowers crowded, floral parts abnormal. Fruits reduced in number, distorted, mottled, with dark green swellings.	Cucumber Mosaic Virus (<i>Cucumis Virus</i> 1), p. 62.

Host Plant	Symptoms	Disease caused by
<i>Cochlearia armoracia</i> , Horseradish.	Leaves curled inwards; exudation of sap from petioles. Later, leaves turn yellow. Roots dwarfed and brittle.	Sugar Beet Curly Top Virus (<i>Beta Virus 1</i>), p. 24.
" "	Plant stunted, yellowed. Leaves stunted with fern-leaf effect, mottled. Old leaves with black elongated lesions. Roots small, scaly, pitted.	Undifferentiated Crucifer Virus, p. 19.
<i>Commelinia nudiflora</i> , Creeping Dayflower.	Leaves mottled with yellowish patches or spots.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 72.
<i>Cosmos bipinnatus</i> . Cosmos.	Internodes shortened, apices of branches and secondary shoots yellow. Leaflets curled and twisted, petioles bent downwards. Flower buds dwarfed, surrounded by clusters of small chlorotic leaves.	Sugar Beet Curly Top Virus (<i>Beta Virus 1</i>), p. 31.
<i>Cucumis melo reticulatus</i> , Muskmelon.	Plant stunted. Young leaves dwarfed, puckered, curled margins. Old leaves yellow. Flowers dwarfed, often dry before petals expand.	Sugar Beet Curly Top Virus (<i>Beta Virus 1</i>), p. 30.
" "	Young leaves turn light yellow and curl downward, and develop mosaic mottle. Old leaves gradually turn yellow, but do not die early. Young fruits mottled with dark green warts. Old fruits nearly normal.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 60.
<i>Cucumis sativus</i> , Cucumber.	Mosaic mottle and rigidity of foliage. Sharp downward or upward bending of end of leaf.	Delphinium Stunt Disease Virus (<i>Delphinium Virus 1</i>), p. 3.
" "	Plant stunted. Young leaves dwarfed, cupped, densely clustered, dark green. Old leaves yellow. Fruit dwarfed, malformed.	Sugar Beet Curly Top Virus (<i>Beta Virus 1</i>), p. 29.
" "	Plant stunted. Young leaves show small light green translucent spots. Older leaves show yellow mottle, distortion, downward curling of margins, wrinkling of surface. Leaf size reduced, internodes and petioles shor-	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 59.

Host Plant	Symptoms	Disease caused by
<i>Cucumis sativus</i> , Cucumber.	tened. Runners few. General habit bunched and bushy, leaves rosetted. Flowers dwarfed and reduced in number. Fruit mottled, distorted, with wart-like protuberances. Occasionally greenish-white misshapen fruits are produced.	
" "	Plant stunted. Young leaves mottled, blistered, distorted. Fruit usually unaffected.	Cucumber Mild Mosaic Virus (<i>Cucumis Virus 2</i>), p. 85.
" "	Plant slightly stunted. Leaves with bright yellow mottle of star-like spots, or vein-banding. Fruit with yellow or silver spots or streaks.	Cucumber Yellow Mosaic Virus (<i>Cucumis Virus 2A</i>), p. 86.
<i>Cucurbita maxima</i> , <i>C. pepo</i> , var. <i>condensata</i> , Squashes.	Young leaves show small brown spots surrounded by bright yellow halos or rings. Young leaves extremely savoyed with dark raised areas, and yellow-green blotches. Fruits mottled with orange-yellow warts.	Tobacco Ringspot Virus (<i>Nicotiana Virus 12</i>), p. 273. Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 62.
<i>Cucurbita pepo</i> , Pumpkin.	Young leaves very mottled and wrinkled. Old leaves show rapid yellowing and wilting. Shoots with shortened internodes and tendency to branch. Fruits mottled and deformed, reduced in number.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 60.
<i>Cucurbita pepo</i> , Pumpkin.	Young leaves dwarfed, cupped, distorted; veins often cleared; discoloration and mottling usually present. Flowers	Sugar Beet Curly Top Virus (<i>Beta Virus 1</i>), p. 29.
<i>Cucurbita maxima</i> , Squash.	dwarfed and tend to drop off. Calyx present, but corolla may not develop.	
<i>Cucurbita moschata</i> , Vegetable Marrow.	Leaves show vein-banding of yellow-green on normal background, becoming less noticeable with age. American varieties may show yellowing and distortion, with shortened internodes and bushy habit.	Dahlia Mosaic Virus (<i>Dahlia Virus 1</i>), p. 208.
<i>Dahlia variabilis</i> , Dahlia.	Flowers usually unaffected.	

Host Plant	Symptoms	Disease caused by
<i>Dahlia variabilis</i> , Dahlia.	Leaves with irregular ring or zigzag markings of yellow-green or pale green, which may become necrotic. No stunting or distortion.	Dahlia Ringspot Virus (<i>Dahlia Virus 2</i>), p. 213.
„ „	Symptoms as in <i>Dahlia Virus 2</i> , but ring and zigzag patterns bright yellow.	Dahlia Yellow Ringspot Virus (<i>Dahlia Virus 2A</i>), p. 213.
„ „	Leaves show pale chlorotic line, like outline of oakleaf. No dwarfing or distortion.	Dahlia Oakleaf Virus (<i>Dahlia Virus 3</i>), p. 214.
„ „	Well-defined mosaic mottle of light or dark green and/or concentric rings and wavy lines on leaves.	Tomato Spotted Wilt Virus (<i>Lycopersicum Virus 3</i>), p. 302.
<i>Datura Stramonium</i>	Leaves with characteristic concentric rings and wavy markings, sometimes an oak-leaf pattern caused by necrosis of the veins.	Tomato Spotted Wilt Virus (<i>Lycopersicum Virus 3</i>), p. 308.
„ „	Small necrotic rings on leaves with considerable necrosis, or alternatively a mosaic mottle, with dark green vein-banding.	Potato Simple Mosaic or X Virus (<i>Solanum Virus 1</i>), p. 347.
„ „	Yellow mosaic, dark green banding of the veins and blistering.	Hyoscyamus Mosaic Virus (<i>Hyoscyamus Virus 1</i>), p. 332.
„ „	Very bold mosaic mottling with patches of white or yellow, leaves blistered and distorted.	Tomato Bushy Stunt Virus (<i>Lycopersicum Virus 4</i>), p. 315.
„ „	Leaves show yellow flecks which become necrotic and coalesce, killing large areas of leaf blades. Leaves remain attached to plant.	(<i>Datura Virus 1</i>), p. 333.
„ „	Leaves show mosaic mottle with chlorotic ring and line patterns. Rings dark green and often concentric.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 66.
<i>Daucus carota</i> , var. <i>sativa</i> , Carrot.	Young leaves yellowed, dwarfed ; petioles twisted. Old leaves reddish or purple. Dense growth of chlorotic shoots sometimes develops at centre of crown.	Aster Yellows Virus (<i>Callistephus Virus 1</i>), p. 217.

Host Plant	Symptoms	Disease caused by
<i>Delphinium</i> sp. Perennial <i>Delphinium</i> .	Stunting ; chlorosis and savoying of leaves ; necroses on leaves and stem ; proliferation of shoots ; lodging of tops ; premature yellowing of foliage and death of shoots at time of flowering.	<i>Delphinium</i> Stunt Disease Virus (<i>Delphinium Virus 1</i>), p. 2.
" "	Chlorotic ring-patterns becoming more prominent with age, with final bleaching of leaves.	(<i>Delphinium Virus 2</i>), p. 5.
" "	Plant chlorotic. Leaves with pale green areas following veins ; faint mosaic mottle.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 55.
<i>Delphinium consolida</i> L., Larkspur.	Plant stunted, chlorotic, flattened and rosetted. Leaflets chlorotic, curled downward, mottled. Flowers rarely produced.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 55.
" "	Plant stunted, chlorotic. Leaves mottled, malformed, with necrotic lesions.	Tobacco Mosaic Virus (<i>Nicotiana Virus 1</i>), p. 236.
" "	Young leaves malformed, edges yellowish, necrotic and inwardly curled, concentric rings sometimes present ; necrotic patches sometimes develop on stems and older leaves, plant somewhat distorted.	Tomato Spotted Wilt Virus (<i>Lycopersicum Virus 3</i>), p. 298.
<i>Emilia sagittata</i> .	Leaves show marked mosaic mottling, frequently of a zonate or ringed character, dark and light green striping on involucres of flower heads.	Pineapple Yellow Spot Virus (<i>Ananas Vir. s 1</i>), p. 401.
<i>Fagopyrum esculentum</i> , Buckwheat.	Plant often killed. Leaves show blister-like elevations ; margins rolled ; petioles twisted. Later, leaves dwarfed and cupped outward.	Sugar Beet Curly Top Virus (<i>Beta Virus 1</i>), p. 25.
" "	Plant stunted. Leaves show severe mottling and malformation.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 55.
" "	Plant slightly stunted and chlorotic, with indefinite proliferation of flower buds and production of small greenish flowers on long pedicels.	Aster yellows Virus (<i>Callistephus Virus 1</i>), p. 216.
" "	Leaves malformed, with pale chlorotic spots and sometimes necrosis.	Tobacco Mosaic Disease Virus (<i>Nicotiana Virus 1</i>), p. 236.

Host Plant	Symptoms	Disease caused by
<i>Ficus carica</i> , Fig.	Leaves with irregular yellow-green blotches, or with pale green spots or bands associated with veins. Little or no distortion. Fruit usually unaffected.	Fig Mosaic Virus (<i>Ficus Virus 1</i>), p. 192.
<i>Foeniculum dulce</i> , Florence Fennel.	Youngest leaves with shortened petioles, leaflets curled, thread-like. Petioles of older leaves drooping.	Sugar Beet Curly Top Virus (<i>Beta Virus 1</i>), p. 31.
<i>Fragaria vesca</i> , Strawberry	Chlorosis or yellowing of the leaves confined to the marginal regions, accompanied by a general dwarfing, irregular curling of the marginal region, downward curling of the mid-rib and twisting of the whole lamina.	Strawberry Yellow-edge Virus (<i>Fragaria Virus 1</i>), p. 102.
" "	Leaves crinkled with chlorosis localised in spots, frequently with small necrotic areas.	Strawberry Crinkle Virus (<i>Fragaria Virus 2</i>), p. 106.
" "	Long, unusually erect, stiff, spindly petioles bearing leaflets smaller than normal. Leaves usually olive green. The mid-veins of the leaflets arch downwards. In some varieties the brooming or bushy character is more pronounced.	Strawberry Witch's Broom Virus (<i>Fragaria Virus 3</i>), p. 108.
" "	Leaves much reduced and strikingly deformed with short petioles and elongated, asymmetrical, crinkled leaflets. The petioles, veins and under surfaces of the leaflets are often reddish-purple. Chlorosis of leaf is absent.	Strawberry Dwarf Disease Virus (<i>Fragaria Virus 4</i>), p. 109.
<i>Freesia</i> sp.	Leaves with small water-soaked areas which later turn to white paper-like spots; these may coalesce and form large necrotic areas; flowers distorted with frequent extension of chlorophyll into the petals.	Freesia Mosaic Virus (<i>Freesia Virus 1</i>), p. 424.
<i>Gesneria</i> sp.	Plant stunted, chlorotic. Leaves with numerous necrotic spots or rings.	Tobacco Mosaic Virus (<i>Nicotiana Virus 1</i>), p. 245.

Host Plant	Symptoms	Disease caused by
<i>Gloxinia</i> sp.	Plant shows slight chlorosis and produces many secondary shoots, and no flowers.	Aster yellows Virus (<i>Callistephus Virus</i> 1), p. 221.
“ ”	Leaves with large single rings, with wide dark edges.	Tomato Spotted Wilt Virus (<i>Lycopersicum Virus</i> 3), p. 308.
<i>Gossypium peruvianum</i> × <i>barbadense</i> , Cotton.	Veins of the leaves thickened, they appear opaque and of a darker green when viewed against the light. Oval cup-like foliar growths may be produced on the lower sides of the primary veins.	Cotton Leaf-curl Virus (<i>Gossypium Virus</i> 1), p. 92.
<i>Helichrysum bracteatum</i> , Strawflower.	Numerous secondary shoots develop near branch tips; older leaves curled outwards with protuberances on distorted veins; old leaves show vein-clearing; young leaves linear, twisted. Flowers dwarfed.	Sugar Beet Curly Top Virus (<i>Beta Virus</i> 1), p. 31.
<i>Hesperis matronalis</i> , Sweet Rocket.	Plant crippled or killed. Young leaves mottled, older leaves crinkled or yellowed. Flowers flecked.	Cabbage Ringspot Virus (<i>Brassica Virus</i> 1), p. 12.
<i>Hibiscus cannabinus</i> Til.	Well-marked net-vein evations (outgrowths), veins extremely thick and gnarled on underside. Leaves remain small and are distinctly curled upwards.	Cotton Leaf-curl Virus (<i>Gossypium Virus</i> 1), p. 90.
<i>Hippeastrum</i> spp.	Leaves with pale yellow or white spots, also some blood-red necrotic spots usually associated with the former, but also running along the leaf edge; death of leaf frequently ensues.	Tomato Spotted Wilt Virus (<i>Lycopersicum Virus</i> 3), p. 311.
<i>Holodiscus discolor</i> , Ocean Spray.	Leaves small and crowded, later turning bronzy-red. Short internodes on stems, several buds at each node producing short spindly laterals.	(<i>Holodiscus Virus</i> 1), p. 127.
<i>Humulus lupulus</i> , Hop.	Leaves with yellow mottle, brittle, curled; margins recurved. Bines brittle, unable to climb, barren. Roots partly killed. Hop-cones often malformed. Disease usually fatal.	Hop Mosaic Virus (<i>Humulus Virus</i> 1), p. 193.

Host Plant	Symptoms	Disease caused by
<i>Humulus lupulus</i> Hop.	Plant weak, with numerous shoots, stunted. Bines short, unable to climb. Leaves curled upwards and inwards, with long terminal lobes. Cones few or absent. Disease rarely fatal.	Nettlehead Disease Virus (<i>Humulus Virus 2</i>), p. 194.
,, "	Primary leaves show pale yellow areas, usually along veins ; margins may be serrated and turned up, with more or less distortion.	Hop Chlorotic Disease Virus (<i>Humulus Virus 3</i>), p. 195.
,, "	Symptoms as in <i>Humulus Virus 1</i> , but disease appears late in season and allows greater growth of bine.	(<i>Humulus Virus 4</i>), p. 196.
<i>Hyacinthus</i> spp., Hyacinth.	Light green or light yellow stripes running longitudinally down the leaves, stripes more pronounced on the upper leaf surface. Flowers poor and abnormally small.	Narcissus Stripe Virus (? <i>Tulipa Virus 1</i>), p. 413.
<i>Hyoscyamus niger</i> , Henbane. ,, "	Plant severely stunted. Leaves show vein-clearing followed by yellowing and bright mottle. Clearing or yellowing of leaf veins, followed by a yellow mosaic and dark green banding of the veins.	Tobacco Mosaic Virus (<i>Nicotiana Virus 1</i>), p. 243.
<i>Iris tingitana</i> , <i>Iris</i> spp., Bulbous Iris.	Mottling and yellowish striping of the leaves, bud sheath marked with bluish-green blotches on a pale green ground ; flowers frequently with "broken" colours.	Hyoscyamus Mosaic Virus (<i>Hyoscyamus Virus 1</i>), p. 331.
<i>Iris tuberosa</i> .	Symptoms similar to above, pale areas on leaves more conspicuous.	Iris Stripe Virus (<i>Iris Virus 1</i>), p. 421.
<i>Lactuca sativa</i> , Lettuce.	Plant dwarfed, chlorotic ; fails to make head, but produces many upright secondary shoots. Leaf margins may show brown pustules. Flowering side-branches shortened.	Aster Yellows Virus (<i>Callistephus Virus 1</i>), p. 220.
,, "	Plant stunted, may be rosetted with no head, yellowed. Young leaves yellowed along veins. Older leaves mottled with	Lettuce Mosaic Disease Virus (<i>Lactuca Virus 1</i>), p. 227.

Host Plant	Symptoms	Disease caused by
<i>Lactuca sativa</i> , Lettuce.	patches or spots of darker green near main veins, abnormally wrinkled. Slight marginal wilting, necrotic spotting and slight yellowing of the leaves ; lateral curvature of many leaves ; diseased plants always lean to one side in contrast to erect healthy heads.	Tomato Spotted Wilt Virus (<i>Lycopersicum Virus 3</i>), p. 304.
<i>Lactuca scariola</i> , Lettuce (Prickly).	Dwarfing, curling and folding of leaf with blotchy mottle of white necrotic areas.	Delphinium Stunt Disease Virus (<i>Delphinium Virus 1</i>), p. 3.
<i>Lilium longiflorum</i> , Easter Lily. <i>L. auratum</i> , Golden Lily. <i>L. candidum</i> .	Leaves mottled, streaked and distorted with dead necrotic spots, flowers small and greenish, or may not develop.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 74.
<i>Lilium longiflorum</i> , var. <i>eximium</i> .	Leaves slightly chlorotic and curled downwards in a marked manner, plant in the form of a flat rosette ; inner scales of bulb tightly drawn together.	Lily Rosette Virus (<i>Lilium Virus 1</i>), p. 416.
<i>Lobelia cardinalis</i> .	Young leaves distorted, twisted ; with patchy mosaic of dark and pale green. Old leaves distorted, brittle. Flower colour unaffected.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 68.
<i>Lunaria annua</i> , Honesty.	Leaves show vein-clearing followed by mottle. In advanced stages plant stunted, leaves distorted.	Cauliflower Mosaic Virus (<i>Brassica Virus 3</i>), p. 16.
,, ,,	Leaves coarsely mottled light green on dark.	(Undifferentiated Crucifer Virus), p. 20.
<i>Lupinus angustifolius</i> , Lupin.	Leaves distorted ; leaflets with necrotic spots ; stem striped longitudinally. Flowers reduced or absent.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 64.
<i>Lupinus leucophyllus</i> .	Necrotic spots and concentric rings on the leaves giving rise to distortion and malformation.	Tomato Spotted Wilt Virus (<i>Lycopersicum Virus 3</i>), p. 300.
<i>Lycopersicum esculentum</i> , Tomato.	Dwarfing ; vein-clearing ; rolling of leaf margins and tips ; faint mottle intensifying with age ; corkscrew distortion of leaves in old plant.	Delphinium Stunt Disease Virus (<i>Delphinium Virus 1</i>), p. 3.

Host Plant	Symptoms	Disease caused by
<i>Lycopersicum esculentum</i> , Tomato.	<i>Field symptoms.</i> Plant erect, rigid ; growth arrested ; stem hollow ; leaves thickened, crisp, yellow ; veins purple ; leaflets rolled inwards, drooping. Fruits ripen prematurely, seeds abortive. Roots decayed. Plant finally killed.	Sugar Beet Curly Top Virus (<i>Beta Virus 1</i>), p. 32.
" "	<i>Glasshouse symptoms.</i> Plant stunted ; leaflets curled inwards ; veins cleared ; white excrescences on veins ; yellowing between veins. Plant finally turns yellow and dies.	Sugar Beet Curly Top Virus (<i>Beta Virus 1</i>), p. 32.
" "	Young leaves spindled, spirally twisted, curved, filiform. Older leaves chlorotic, often rolled or folded. Numerous lateral leaflets.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 69.
" "	Plant dwarfed, chlorotic, bushy. Many secondary shoots, and stimulation of growth in axils of leaves and leaflets.	Aster Yellows Virus (<i>Callistephus Virus 1</i>), p. 221.
" "	Plant normal or stunted. Leaves show bright mottle with raised dark green areas ; distortion ; sometimes fern-leaf. Fruit unaffected.	Tomato Mosaic Virus (<i>Nicotiana Virus 1</i>), p. 244.
" "	Plant overgrown. Young leaves mottled. Old leaves thread-like, or with many very small leaflets and "corkscrew" tips, or with outgrowths from under surface (enations). Fruit normal or conical and malformed, of poor quality.	Tobacco Distorting (Enation) Virus (<i>Nicotiana Virus 1A</i>), p. 247.
" "	Young leaves with bright mottle ; some distortion. Older leaves very dark green with light mottling, later turn yellow with dark vein-banding. Necrosis may be present. Fruit mottled with yellow or pale green.	Tomato Yellow Leaf Virus (<i>Nicotiana Virus 1B</i>), p. 249.
" "	Plant stunted, of spindling habit. Young leaves curled downwards, wrinkled, with chlorotic spots. Older leaves pale yellow with dark green blisters. Fruit normal or mottled.	Tomato Aucuba Mosaic Virus (<i>Nicotiana Virus 1C</i>), p. 251.

Host Plant	Symptoms	Disease caused by
<i>Lycopersicum esculentum</i> , Tomato.	Stems show dark streaks of varying length; leaves show necrotic spots and patches; fruit with irregular sunken blotches.	Tomato Streak Virus (<i>Lycopersicum Virus 1</i>), p. 290.
" "	Leaves show necrotic spots; veins streaked; leaves drooping or dead. Streaks spreading to stem. Yellow oak-leaf patterns may develop between leaf veins. Fruits show chlorotic sunken rings.	Tomato Ring Mosaic Streak Virus (<i>Lycopersicum Virus 2</i>), p. 293.
" "	Young leaves with a tendency to curl downwards, characteristic bronze-coloured circular markings which tend to coalesce; later, a yellowish mottling, with some leaf distortion.	Tomato Spotted Wilt Virus (<i>Lycopersicum Virus 3</i>), p. 304.
" "	Yellow spots on topmost leaves, characteristic yellow and purple coloration of lower leaves, some concentric rings frequently present; growing point sometimes killed, followed by growth of secondary shoots giving characteristic bushy growth, stem lesion sometimes present at soil level.	Tomato Bushy Stunt Virus (<i>Lycopersicum Virus 4</i>), p. 319.
" "	Leaves small and clustered, later forming dense rosettes, youngest fruit truss in an upright position, calyx enlarged to form a bladder with a toothed opening at the top, purple colour generally present along the veins of these bladder-like calyces; fruit hard and tough, and colours very slowly.	Tomato Big Bud Virus (<i>Lycopersicum Virus 5</i>), p. 324.
" "	Leaves puckered, twisted downwards, reduced in size; necrosis of leaves and stems; plants, if infected early, composed of thin upright axillary branches; if infected late, are of weak habit with thick semi-procumbent stems clothed with numbers of short axillary shoots bearing characteristic small leaves.	Tomato Bunchy Top Virus (<i>Lycopersicum Virus 6</i>), p. 326.

Host Plant	Symptoms	Disease caused by
<i>Lycopersicum esculentum</i> , Tomato.	Young leaves with small circular brown lesions. Growing points killed when plant infected young. In old-infected plants systemic symptoms do not develop.	(<i>Datura Virus</i> 1), p. 335.
" "	Young leaves show necrotic lesions followed by mosaic mottle and occasionally necrotic rings.	Potato Virus X (<i>Solanum Virus</i> 1), p. 346.
<i>Malcomia maritima</i> , Virginia Stock.	Plant stunted, slightly chlorotic, with numerous secondary shoots. Flowers diseased, pistils giving rise to long stems bearing secondary flowers.	Aster Yellows Virus (<i>Callistephus Virus</i> 1), p. 216.
<i>Manihot utilissima</i> , Cassava.	Plant stunted, leaves small with asymmetrical lobes. Characteristic yellow spots on upper surface of leaf which also shows wart-like lesions.	Cassava Mosaic Virus (<i>Manihot Virus</i> 1), p. 95.
" "	Dark brown stripes on otherwise green stem, and yellow mottling of the leaves which only develops on the ageing, and not on the young, leaves.	Cassava Stem Lesion Virus (<i>Manihot Virus</i> 2), p. 96.
<i>Martynia louisiana</i> .	Plants stunted. Young leaves curled, mottled. Fruits dwarfed, rarely mottled with wart-like swellings.	Cucumber Mosaic Virus (<i>Cucumis Virus</i> 1), p. 72.
<i>Matthiola incana</i> , Stock.	Variegated, "broken" flowers. Leaves crinkled with twisted edges, lower leaves chlorotic, upper mottled.	Cabbage Ringspot Virus (<i>Brassica Virus</i> 1), p. 10.
" "	Leaves with dark green mottle and wavy margins. Flowers unaffected.	Turnip Mosaic Virus (<i>Brassica Virus</i> 2), p. 18.
" "	Plant rosetted. Leaves show vein-clearing and upward curling of margins. Flowers unaffected.	Cauliflower Mosaic Virus (<i>Brassica Virus</i> 3), p. 16.
" "	Plant stunted. Leaves mottled; no necrosis. Flowers "broken." Pods reduced in size.	Stock Mosaic Virus (<i>Matthiola Virus</i> 1), p. 18.
" "	Plant stunted with numerous axillary shoots. Leaves twisted, margins curled, apices often purple or yellow. Veins distorted with wart-like protuber-	Sugar Beet Curly Top Virus (<i>Beta Virus</i> 1), p. 28.

Host Plant	Symptoms	Disease caused by
<i>Medicago hispida</i> , Bur Clover.	ances. Lower leaves dry, discoloured. Brown liquid exudes from leaves and stem. Flowers often malformed.	
<i>Medicago sativa</i> , Alfalfa, Lucerne.	The three leaflets fold along distorted mid-rib ; youngest leaves show vein-clearing. Leaves with greenish-yellow spots frequently in the form of rings, colour later changes to yellow or almost white and runs parallel to the veins and frequently involves them. Leaf tissue is brittle and thickened, breaking easily when bent.	Sugar Beet Curly Top Virus (<i>Beta Virus 1</i>), p. 30. Lucerne or Alfalfa Mosaic Virus (<i>Medicago Virus 1</i>), p. 178.
„ „	Shortening of stems and reduction in size of leaves, blossoming retarded or inhibited, plant greatly dwarfed.	Lucerne or Alfalfa Dwarf Virus (<i>Medicago Virus 3</i>), p. 181.
„ „	Leaves reduced in size, more rounded than normal, frequently exhibiting marginal chlorosis, puckering and distortion of laminæ. Diseased plants produce a thick, dense mass of shoots not exceeding a few inches in height, with an erect bunched appearance. Flowers usually absent, but, if present, are abnormal and distorted.	Lucerne or Alfalfa Witch's Broom Virus (<i>Medicago Virus 4</i>), p. 182.
<i>Melilotus alba</i> , White Sweet Clover.	Youngest leaflets cupped outwards along mid-rib ; faint vein-clearing. Similar symptoms in <i>M. indica</i> ; <i>Trifolium repens</i> ; <i>T. hybridum</i> ; <i>T. incernatum</i> ; <i>T. pratense</i> .	Sugar Beet Curly Top Virus (<i>Beta Virus 1</i>), p. 31.
<i>Melilotus officinalis</i> , Yellow Sweet Clover.	Plant dwarfed. Leaves yellowed, showing light coloured areas and faint rings.	Tobacco Ringspot Virus (<i>Nicotiana Virus 12</i>), p. 275.
<i>Micromelotis lobata</i> , Wild Cucumber.	Leaves deformed ; variegated with light yellow areas contrasting with deep green raised areas ; wrinkled, curled. Fruits irregular with wart-like swellings and split skin.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 63.

Host Plant	Symptoms	Disease caused by
<i>Musa cavendishii</i> , Cavendish Banana.	Dark green streaks along the secondary veins on the under-side of the leaf blade, followed by the development of erect leaves bunched together at the apex of the plant to form a rosette.	Banana Bunchy Top Virus (<i>Musa Virus 1</i>), p. 405.
.. ..	Severe yellowing and mottling of the younger foliage, followed by a rotting of the heart leaf and central portion of the pseudo-stem.	Banana Mosaic Virus (<i>Musa Virus 3</i>), p. 410.
<i>Musa sapientum</i> , Banana.	Plant stunted, rosetted. Leaves chlorotic, drooping, slow-growing, brittle; petioles chlorotic, spotted, reduced.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 75.
<i>Musa textilis</i> , Manila Hemp.	Leaves reduced, narrow; leaf sheaths dark green and shortened to produce congestion and rosetting at the crown; root system degenerated.	Manila Hemp Bunchy Top Virus (<i>Musa Virus 2</i>), p. 408.
<i>Narcissus</i> spp.	Light green or light yellow stripes running longitudinally down the leaves. Flowers poor and abnormally small.	Narcissus Stripe Virus (? <i>Tulipa Virus 1</i>), p. 414.
<i>Nasturtium officinale</i> , Watercress.	Plant stunted. Leaves deformed with yellow mottle.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 55.
<i>Nicandra physaloides</i> , Apple of Peru.	Water-soaked areas coalescing into large brown necrotic lesions often involving entire leaf. Dwarfsing.	Delphinium Stunt Disease Virus (<i>Delphinium Virus 1</i>), p. 4.
.. ..	Plant stunted with small leaves and numerous short lateral branches.	Tomato Bunchy Top Virus (<i>Lycopersicum Virus 6</i>), p. 329.
<i>Nicotiana glutinosa</i> .	Plant stunted. Leaves may show necrosis and severe mottling, occasionally with dark green blisters.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 68.
.. ..	Necrosis along the fine veins of youngest leaves, cessation of growth, centre leaves pallid with margins bending downwards, progressive necrosis involving death of plant.	Tomato Spotted Wilt Virus (<i>Lycopersicum Virus 3</i>), p. 306.

Host Plant	Symptoms	Disease caused by
<i>Nicotiana tabacum</i> , Tobacco.	Inoculated leaves show pale green circular spots after two to three days (no necrosis). Systemic infection; vein-clearing followed by general mottle, sometimes distortion or narrowing of leaves.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 68.
" "	Young leaves show vein-clearing followed by distortion, production of dark green blisters and raised or sunken yellow areas. Lamina may be much reduced. High temperatures produce necrosis and tissue destruction, and possibly partial sterility.	Tobacco Mosaic Virus (<i>Nicotiana Virus 1</i>), p. 239.
" "	Stems and leaf veins show brown or grey sunken stripes. Leaves very crumpled and deformed.	Tobacco Stripe and Curl Virus (<i>Nicotiana Virus 5</i>), p. 257.
" "	Leaves slightly stunted or deformed; faint green mottle developing into intense mosaic of light and dark green areas.	Tobacco Green Mosaic Virus (<i>Nicotiana Virus 6</i>), p. 258.
" "	Leaves show small chlorotic spots with necrotic margins. Older leaves with yellowish necrotic veins.	Tobacco Etch Virus (<i>Nicotiana Virus 7</i>), p. 259.
" "	Leaves show necrotic spots, lines and circles, with vein-clearing. No mottling or chlorosis.	Tobacco Streak Virus (<i>Nicotiana Virus 8</i>), p. 260.
" "	Plant often stunted, stem necrotic and black. Young leaves show yellow areas with brown necrotic spots. Leaves blistered, yellow, drooping, margins irregular. Plant frequently killed.	Tobacco Rotterdam B Disease Virus (<i>Nicotiana Virus 9</i>), p. 261.
" "	Plant stunted. Leaves twisted, curled, with leafy outgrowths on veins of under surfaces. Chlorosis and necrosis absent.	Tobacco Leaf-curl Virus (<i>Nicotiana Virus 10</i>), p. 262.
" "	Seedlings show on lowest leaves necrosis which spreads and kills plant. Older plants show necrosis on lower leaves which dry up, but rest of plant remains unaffected. No chlorosis.	Tobacco Necrosis Virus (<i>Nicotiana Virus 11</i>), p. 268.

Host Plant	Symptoms	Disease caused by
<i>Nicotiana tabacum</i> , Tobacco.	Leaves show concentric rings with necrotic centres. Necrosis sometimes follows mid-rib and longer veins. Plants tend to show apparent recovery. (See also <i>Nicotiana Virus 12A</i> , 12B, and 13.)	Tobacco Ringspot Virus (<i>Nicotiana Virus 12</i>), p. 277.
" "	Young leaves show general mottling and incomplete chlorotic rings and some necrotic rings. Sometimes vein-banding. Plants show apparent recovery.	Bergerac Ringspot Virus (<i>Nicotiana Virus 14</i>), p. 286.
" "	Leaves show necrotic lesions sometimes coalescing and spreading down petiole to stem, which becomes severely affected and may cause collapse of plant.	Tomato Streak Virus (<i>Lycopersicum Virus 1</i>), p. 292.
" "	Zoned spots on leaves and large or small concentric rings; under certain conditions large scorch-like necroses develop, involving rapid death of plant.	Tomato Spotted Wilt Virus (<i>Lycopersicum Virus 3</i>), p. 305.
" "	Leaves show preliminary clearing of veins, followed by yellow mosaic with broad, blistered, dark green bands; later leaves exhibit a confused chequered design of dark bands, yellow mottle and necrotic spots.	Hyoscyamus Mosaic Virus (<i>Hyoscyamus Virus 1</i>), p. 332.
" "	Young leaves show vein-clearing and numerous necrotic spots. Older leaves symptomless, but may break out into oak-leaf or necrotic ring patterns.	(<i>Datura Virus 1</i>), p. 333.
" "	Necrotic rings and mottle.	Potato Virus X (<i>Solanum Virus 1</i>), p. 347.
" "	Clearing of the veins followed by banding.	Potato Virus Y (<i>Solanum Virus 2</i>), p. 349.
<i>Oryza sativa</i> , Rice Plant.	Yellowish-white specks on leaves, specks elongate and spread out along the leaf parallel to the mid-rib, forming fine interrupted streaks; growth is arrested and the plant becomes stunted, with shortened internodes,	Dwarf or Stunt Virus (<i>Oryza Virus 1</i>), p. 458.

Host Plant	Symptoms	Disease caused by
<i>Oryza sativa</i> , Rice Plant.	numerous tillers develop, producing a rosette appearance. Leaves with pronounced yellow stripes, plant stunted.	Rice Stripe Virus (<i>Oryza Virus 2</i>), p. 454.
<i>Paeonia</i> sp., Peony.	Pronounced yellow mosaic or chlorotic rings, occasionally small necrotic spots.	Peony Ringspot Virus (<i>Paeonia Virus 1</i>), p. 5.
<i>Papaver nudicaule</i> , Poppy.	Plant severely stunted. Chlorotic.	Aster Yellows Virus (<i>Callistephus Virus 1</i>), p. 216.
" "	Leaves twisted and yellow, centre of plant with a pallid stunted appearance; purplish blotches may develop on the yellowed leaves or on their mid-ribs and petioles, petiole frequently necrosed; flowers deformed.	Tomato Spotted Wilt Virus (<i>Lycopersicum Virus 3</i>), p. 298.
<i>Passiflora edulis</i> , Passion Fruit.	Plants appear stunted. Stems with mottled dark green areas. Leaves stunted, curled, crinkled, with yellow-green chlorosis or mosaic. Fruits undersized, deformed, with thickened rind.	Passion Fruit Bullet Disease Virus (<i>Passiflora Virus 1</i>), p. 50.
<i>Pastinaca sativa</i> , Parsnip.	Plant stunted, chlorotic, with many secondary shoots. Leaves small; petioles twisted.	Aster Yellows Virus (<i>Callistephus Virus 1</i>), p. 27.
<i>Pelargonium hortorum</i> , Geranium.	Young leaves cupped inward, with sinuous cleared veins, protuberances on lower surfaces. Old leaves chlorotic with green vein-banding. Older branches yellow.	Sugar beet Curly Top Virus (<i>Beta Virus 1</i>), p. 28.
" "	Plant stunted. Leaves mottled.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 57.
<i>Pelargonium zonale</i> , <i>P. hederaceum</i> .	Severely affected plants malformed. Normally the young leaves show yellow chlorotic spots or star-shaped blotches, sometimes with vein-clearing. Later the spots become necrotic, turning brown. Older leaves crinkled, split. Affected plants frequently show apparent recovery.	Pelargonium Leaf-curl Virus (<i>Pelargonium Virus 1</i>), p. 49.

Host Plant	Symptoms	Disease caused by
<i>Penstemon</i> sp.	Leaves slightly mottled, chlorotic, some twisting and malformation.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 72.
<i>Petroselinum hortense</i> , var. <i>radicosum</i> .	Dense growth of chlorotic leaves at centre of crown. Leaves dwarfed ; petioles upright, twisted.	Aster Yellows Virus (<i>Callistephus Virus 1</i>), p. 217.
Turnip-rooted Parsley.		
<i>Petunia hybrida</i> ,	Plant stunted. Numerous secondary shoots. Leaves dwarfed, cupped. Veins with protuberances on lower surfaces. Corolla often fails to expand and becomes dry. Final yellowing and death of plant.	Sugar Beet Curly Top Virus (<i>Beta Virus 1</i>), p. 33.
Petunia.		
„ „	Stem internodes short, sometimes with necrotic streaks. Leaves mottled, necrotic ; stunted. Flowers undeveloped, or mottled with streaks and rings.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 70.
„ „	Plant dwarfed, chlorotic. Secondary shoots much branched, rosetted. Flowers deformed.	Aster Yellows Virus (<i>Callistephus Virus 1</i>), p. 221.
„ „	Young leaves cupped, older leaves distorted, mottled.	Tobacco Mosaic Virus (<i>Nicotiana Virus 1</i>), p. 243.
„ „	Seedlings severely stunted. Leaves mottled, streaked, with slight necrosis. Margins curled. Older plants may show partial "recovery." Seeds reduced in number.	Tobacco Ringspot Virus (<i>Nicotiana Virus 12</i>), p. 278.
<i>Phaseolus vulgaris</i> , French Bean.	Young leaves dwarfed, puckered, cupped outward, dark green ; veins cleared. Early infected plants usually bear no pods.	Curly Top Virus (<i>Beta Virus 1</i>), p. 30.
„ „	Mosaic pattern of light and dark green areas on the leaves accompanied by blistering and marginal curling ; proliferation of the veins, stunting of the plant and deformation of pods and flowers.	Common Bean Mosaic Virus (<i>Phaseolus Virus 1</i>), p. 157.
„ „	Leaflets curled downwards and pointing downwards from place of attachment, surface slightly irregular with distinct mottling of yellow and dark green areas.	Yellow Bean Mosaic Virus (<i>Phaseolus Virus 2</i>), p. 161.

Host Plant	Symptoms	Disease caused by
<i>Phaseolus vulgaris</i> , French Bean.	Primary leaves show small round lesions, which remain localised. Sometimes severe systemic necrosis develops and leaf shrivels and dies, followed by stem necrosis and death of plant.	(<i>Datura Virus 1</i>), p. 333.
<i>Physalis peruviana</i> , Cape Gooseberry.	Severe stunting, reduction in leaf size and bunching of foliage ; followed by development of numerous short axillary shoots and the dying of the older leaves.	Tomato Bunchy Top Virus (<i>Lycopersicum Virus 6</i>), p. 329.
<i>Physalis viscosa</i> .	Plant stunted, much branched and bearing small leaves.	Tomato Bunchy Top Virus (<i>Lycopersicum Virus 6</i>), p. 328.
<i>Phytolacca americana</i> , Pokeweed.	Young leaves with green-yellow mottle ; curled downward. Old leaves wrinkled, blistered.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 56.
<i>Pisum sativum</i> , Pea.	Leaves mottled, crinkled and savoyed with proliferations (enations) on the undersides ; pods distorted, ridged and wrinkled.	Enation Pea Mosaic Virus (<i>Pisum Virus 1</i>), p. 167.
“ ”	Leaves with distinct mottle of yellow and green patterns with yellow streaks between the veins, wrinkled, puckered, twisted and smaller than normal.	Common Pea Mosaic Virus (<i>Pisum Virus 2</i>), p. 169.
“ ”	Streaking and necrotic spots on pods, stems and leaves ; pods may show necrotic circular pitting or they may collapse entirely.	Pineapple Yellow Spot Virus (<i>Ananas Virus 1</i>), p. 404.
“ ”	Leaves occasionally mottled, purplish streak on stem ; necrotic spots or concentric patterns on pods.	Tomato Spotted Wilt Virus (<i>Lycopersicum Virus 3</i>), p. 300.
<i>Plantago major</i> , Greater Plantain.	Necrotic rings or spots on leaves, some necrosis of the petioles.	Tomato Spotted Wilt Virus (<i>Lycopersicum Virus 3</i>), p. 295.
<i>Primula malacoides</i> .	Yellowing of leaves, withering and marked stunting ; diseased plants usually die.	Tomato Spotted Wilt Virus (<i>Lycopersicum Virus 3</i>), p. 304.

Host Plant	Symptoms	Disease caused by
<i>Primula obconica.</i>	Plant very stunted. Young leaves small, distorted, yellow-mottled; retarded growth. Flowers may show flecks or streaks on petals.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 68.
<i>Primula Sinensis.</i>		
<i>Primula Sinensis.</i>	Irregular necrotic lesions on leaves which later form scorch-like patches, veins of younger leaves prominent and slightly yellow; plant stunted.	Tomato Spotted Wilt Virus (<i>Lycopersicum Virus 3</i>), p. 304.
<i>Prunus armeniaca,</i> Apricot.	Faint mottling of leaves which are sprinkled with lighter green spots, blotches and occasionally rings.	Peach Mosaic Virus (<i>Prunus Virus 5</i>), p. 145.
<i>Prunus avium,</i> Sweet Cherry.	Ringspot type of mottle on leaves or streaks along the base of the mid-rib or side veins of the leaves; sometimes several distinct rings of lighter green are present.	Peach Mosaic Virus (<i>Prunus Virus 5</i>), p. 147.
<i>Prunus communis,</i> Almond.	Leaves with rings or mosaic mottle, fruits bumpy and irregularly developed.	Peach Mosaic Virus (<i>Prunus Virus 5</i>), p. 146.
<i>Prunus domestica,</i> Plum.	Leaves mottled with light green or yellowish-green blotches which may be ring-like or in the form of streaks.	Peach Mosaic Virus (<i>Prunus Virus 5</i>), p. 146.
<i>Prunus persica,</i> Peach.	Presence of "witches' brooms," premature ripening of the fruit, premature unfolding of leaf buds, tendency of the terminal shoots of larger limbs to grow vertically. Shoots of a slender, wiry character.	Peach Yellows Virus (<i>Prunus Virus 1</i>), p. 129.
" "	Affected trees are stunted, with pale yellow leaves and compact bushy appearance. Foliage deeper green than usual, fruit small and late-ripening.	Little Peach Virus (<i>Prunus Virus 1A</i>) p. 188.
" "	Compact tufts of rosettes, 2 to 8 inches long, containing several hundred leaves. At the base of these tufts are one or two abnormally long and straight leaves with inrolled margins. Affected trees either wilt and die or develop the characteristic rosette type of growth.	Peach Rosette Virus (<i>Prunus Virus 2</i>), p. 185.

Host Plant	Symptoms	Disease caused by
<i>Prunus persica</i> , Peach.	Shortened internodes with a large number of lateral twigs and flattened dark green leaves giving the appearance of compact dense growth with luxuriant foliage. Fruit much dwarfed but otherwise normal in appearance.	Phony Disease Virus (<i>Prunus Virus 3</i>), p. 138.
" "	At ripening time, a yellowish-green or bronzed appearance of the tree. Leaf clusters often sprout from most of the buds and an unusual number of short shoots arise along the main branches. The fruit frequently has a bumpy or rough contour and shows an abnormal deep red or purple blush on the exposed side.	Red Suture Virus (<i>Prunus Virus 4</i>), p. 141.
" "	Marked dwarfing in, and profuse branching of, tree; leaves with conspicuous yellow mottling and crinkling. Fruit smaller than normal, irregular in shape and as a rule bumpy along the suture.	Peach Mosaic Virus (<i>Prunus Virus 5</i>), p. 143.
<i>Prunus</i> sp. Prune.	Leaves small and narrow, with rugosity and mottling. Leaf blade thickened and margin very irregular in outline. Leaves reduced in size, narrow in proportion to length; serration and pubescence suppressed with considerable rugosity and mottling. Leaf margins extremely irregular in outline, surfaces somewhat glazed.	(<i>Prunus Virus 6</i>), p. 149.
<i>Pyrus</i> <i>aucuparia</i> , Mountain Ash.	Tips of leaves at first yellow, later white, a mottle of yellow spots sometimes present, chlorotic tissue turns white and then brown.	(<i>Pyrus Virus 1</i>), p. 150.
<i>Pyrus</i> <i>communis</i> , Pear.	Chlorosis of the leaves; superficial, ill-defined spots on pedicels, at first pale, these spots later turn blackish-brown, followed by shedding of the young fruits.	Apple Mosaic Virus (<i>Pyrus Virus 2</i>), p. 151.

Host Plant	Symptoms	Disease caused by
<i>Pyrus cydonia</i> , Quince.	Distortion and malformation of leaves, accompanied by scorching and chlorosis; fruit deformed and furrowed.	Apple Mosaic Virus (<i>Pyrus Virus</i> 2), p. 151.
<i>Pyrus malus</i> , Apple.	Variable mottling on leaves, small irregular yellow spots or pale green polygonal spots; occasional slight deformation of fruit.	Apple Mosaic Virus (<i>Pyrus Virus</i> 2), p. 150.
<i>Radicula sylvestris</i> , Druse.	Plant dwarfed, with many chlorotic secondary shoots.	Aster Yellows Virus (<i>Callistephus Virus</i> 1), p. 216. (Undifferentiated Crucifer Virus), p. 20.
<i>Raphanus sativus</i> , Radish. " "	Leaves mottled with patches of dark green near veins. Plant stunted. Leaves yellowed, curled with prominent veins.	(Undifferentiated Crucifer Virus), p. 20. Sugar Beet Curly Top Virus (<i>Beta Virus</i> 1), p. 24. (Undifferentiated Crucifer Virus), p. 20.
<i>Raphanus</i> sp. Chinese Radish.	Leaves coarsely mottled with raised dark green islands; distorted.	(Undifferentiated Crucifer Virus), p. 20.
<i>Rheum raponticum</i> , Rhubarb.	Pale chlorotic areas on leaves, plant stunted.	(Undifferentiated Rhubarb Virus), p. 553.
<i>Ribes nigrum</i> , Black Currant.	Leaves small, narrow and flattened at the base. Surface of leaf coarsely rugose instead of relatively smooth. Less than five submain veins present.	Reversion, (<i>Ribes Virus</i> 1) p. 97.
<i>Richardia africana</i> , Arum Lily.	Leaves somewhat twisted, numerous whitish spots sometimes ring-like on surface; white streaks on leaf and flower stalks and pale blotchy spots on green flower buds; flowers deformed.	Tomato Spotted Wilt Virus (<i>Lycopersicum Virus</i> 3), p. 311.
<i>Rosa</i> sp. Hybrid Tea Rose.	Leaflets with distinct chlorotic areas, puckered and ruffled, plant dwarfed in all parts including roots; buds often bleached.	Rose Mosaic Virus (<i>Rosa Virus</i> 1), p. 152.
" "	Bright yellow mosaic mottling on leaves.	Rose Yellow Mosaic Virus (<i>Rosa Virus</i> 2), p. 154.

Host Plant	Symptoms	Disease caused by
<i>Rosa</i> , sp. Hybrid Tea Rose.	Leaves recurved and crowded on shoots, young stems discoloured and dying back. Characteristic translucent yellowish-green appearance on the young stem. Defoliation from tip of stem downwards.	Rose Wilt or Dieback Virus (<i>Rosa Virus</i> 3), p. 154.
" "	Reddish patterns of rings and vein-banding on the leaves, ring patterns on stem.	Rose Streak Virus (<i>Rosa Virus</i> 4), p. 155.
<i>Rosa manetti</i> .	Numerous minute chlorotic areas distributed over the entire leaflet or concentrated towards the tip. Alternatively a more general chlorosis giving a typical mosaic-like mottle.	Rose Mosaic Virus (<i>Rosa Virus</i> 1), p. 153.
<i>Robinia</i> <i>pseudacacia</i> , Black Locust.	Proliferation and reduction of shoots and leaves, erect "witch's brooms"; "cushions" bearing groups of buds and tiny leaves sometimes present on trunk.	Brooming Disease Virus (<i>Robinia</i> <i>Virus</i> 1), p. 184.
<i>Rubus idaeus</i> , Raspberry.	Mild or distinct mottling, leaves often deeper green than normal. Variable amount of dwarfing of plant. In some varieties leaf petioles and cane tips necrotic and brittle.	Raspberry Green Mosaic Virus (<i>Rubus Virus</i> 1), p. 114
" "	Plant stunted, with leaves decidedly yellow. In black raspberries the leaflets may be longer and narrower than usual, curled and distorted. Most of the area of the leaflet is whitish or yellow.	Raspberry Yellow Mosaic Virus (<i>Rubus Virus</i> 2), p. 115.
" "	Curling of the leaves, deep green foliage and stunting of entire plant. Leaves with a characteristic greasy appearance and a bronze coloration in late summer.	Raspberry Curl Disease Virus (<i>Rubus Viruses</i> 3 and 3 A), p. 122.
" "	Leaves distinctly curled, of a darker green and placed close together on the canes. The leaves have a peculiar twist of the mid-rib. Bluish-violet streaks on the stems; leaves not mottled.	Raspberry Streak Disease Virus (<i>Rubus Virus</i> 4), p. 123.

Host Plant	Symptoms	Disease caused by
<i>Rubus fruticosus</i> , Blackberry.	Leaves smaller than normal, obovate in shape, stems with typical foreshortening and spindling growth. Leaves usually crinkled with a chlorotic mottling which consists of a finely netted spotting.	Blackberry Dwarf Disease Virus (<i>Rubus Virus 5</i>), p. 124.
<i>Saccharum officinarum</i> L., Sugar Cane.	Leaves with pale patches or blotches in the green tissue, blotches irregularly oval or oblong in outline, their longer axes lying parallel to mid-rib.	Sugar Cane Mosaic Virus (<i>Saccharum Virus 1</i>), p. 427.
" "	Leaves with elongated swellings or galls on the under surface; the last leaves unfolding from the spindle shortened and crumpled.	Fiji Disease Virus (<i>Saccharum Virus 2</i>), p. 434.
" "	Plant unable to grow, majority of shoots short and stunted, vascular bundles in the sticks coloured red; pronounced growth of adventitious roots under the leaf sheaths.	Sereh Disease Virus (<i>Saccharum Virus 3</i>), p. 436.
" "	Leaves erect, stunted and clustered, streaked with yellow, tips reddish; cessation of growth.	Dwarf Disease Virus (<i>Saccharum Virus 5</i>), p. 437.
" "	Leaves exhibit a pattern of broken, narrow, pale stripes running in the same direction as the veins; each stripe is uniform and measures $\frac{1}{2}$ mm. in width, in length it varies from $\frac{1}{2}$ mm. to a centimetre or more.	Maize Streak Virus (<i>Zea Virus 2</i>), p. 444.
<i>Santalum album</i> , Sandal.	Stem internodes short; leaves reduced in size, pale green, crowded, standing out stiffly. Some parts of tree show continuous growth, producing "witch's broom" effect. Flowers rare, or replaced by leaf-like structures. Haustoria and root ends killed, resulting in death of tree.	Sandal Spike Disease Virus (<i>Santalum Virus 1</i>), p. 197.
" "	Symptoms similar to those caused by <i>Santalum Virus 1</i> , but infected shoots longer and drooping, excessive branching absent.	Pendulous Spike Disease Virus (<i>Santalum Virus 1A</i>), p. 200.

Host Plant	Symptoms	Disease caused by
<i>Santalum album</i> , Sandal.	Flowers abnormal, sterile buds reduced in size. Pedicels elongated. Anthers reddish. Pistil swollen, bent. Root-ends and haustoria not killed.	
" "	1st stage. Small branches and leaves drooping. Leaves slightly rolled, with mosaic spots. Leaves not reduced in size. Flowers and fruit unaffected.	Leaf-curl Mosaic Virus (<i>Santalum Virus 2</i>), p. 201.
<i>Secale cereale</i> , Rye.	2nd stage. Leaves wrinkled, mottled; edges ruffled. Twigs dwarfed. Young leaves reduced in size, curled, pale yellow-green. Older leaves thickened, brittle.	
<i>Sicyos angulatus</i> .	Mottling and rosetting similar to that on <i>Triticum sativum</i> .	Wheat Rosette or Mosaic Virus (<i>Triticum Virus 1</i>), p. 449.
<i>Soja max</i> , Soya Bean.	Symptoms similar to those on <i>Micrampelis lobata</i> . Old leaves tend to yellow and die.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 63.
<i>Solanum capsicastrum</i> , Winter Cherry.	Leaflets stunted, misshapen, puckered with dark green puffy areas along veins; plants stunted and petioles and internodes somewhat shortened; pods stunted, flattened and more acutely curved than normal.	Soya Bean Mosaic Virus (<i>Soja Virus 1</i>), p. 165.
<i>Solanum melongena</i> , Egg Plant.	Numerous concentric rings on leaves, some necrosis of the veins.	Tomato Spotted Wilt Virus (<i>Lycopersicum Virus 3</i>), p. 306.
<i>Solanum nigrum</i> , Black Nightshade.	Necrotic spots with light centre, spots enlarge and coalesce, killing a large portion of the leaf; plant usually killed.	Tobacco Ringspot Virus (<i>Nicotiana Virus 12</i>), p. 279.
" "	Light and dark green mottle.	Delphinium Sun Disease Virus (<i>Delphinium Virus 1</i>), p. 4.
" "	Necrotic rings or spots and mottle.	Potato Virus X (<i>Solanum Virus 1</i>), p. 348.
	Faint vein banding which latter disappears.	Potato Virus Y (<i>Solanum Virus 2</i>), p. 352.

Host Plant	Symptoms	Disease caused by
<i>Solanum tuberosum</i> . Potato.	Plant stunted. Leaflets rolled inwards, yellowish, petioles bent. Dwarfed shoots develop near tip of plant. Final yellowing and death of plant.	Sugar Beet Curly Top Virus (<i>Beta Virus 1</i>), p. 32.
" "	Plant develops purple sprouts bearing purple sessile aerial tubers with dwarfed leaves. Leaves brittle, margins rolled inwards, petioles curved downwards. Later, leaves become yellow and dry.	Californian Aster Yellows Virus (<i>Callistephus Virus 1A</i>), p. 222.
" "	Interveinal mottling on leaves, little or no dwarfing of plant or deformation of foliage; on some varieties an acute necrosis of the growing points (top necrosis); sometimes necrosis, with cork formation, in the tubers.	Simple Mosaic or X Virus (<i>Solanum Virus 1</i>), p. 344.
" "	In potato President, blotchy mottle on topmost leaves; later fine necroses develop along the veins on undersides of leaves, followed by destruction of the middle leaves, which remain attached to the stem; necrosis of the stem and petioles also present.	Leaf-drop Streak or Y Virus (<i>Solanum Virus 2</i>), p. 349.
" "	Faint, fleeting mosaic mottle without distortion, in the varieties, Irish Chieftain, President, Arran Victory and Epicure. A severe top necrosis disease involving death of the plant in the following varieties: British Queen, Up-to-Date, International Kidney, Kerr's Pink, Rhoderick Dhu, Sharpe's Express and Great Scot; necrosis of tubers also present.	Potato Virus A (<i>Solanum Virus 3</i>), p. 355.
" "	Necrotic spots on youngest topmost leaves, growing points killed in varieties Arran Victory and President, probably also in many other varieties, such as the Arrans, British Queen, Golden Wonder, Kerr's Pink and Sharpe's Express.	Up-to-Date Streak Virus (<i>Solanum Virus 4</i>), p. 358.

Host Plant	Symptoms	Disease caused by
<i>Solanum tuberosum</i> , Potato.	Severe top necrosis, streaking of the stem and petioles, numerous small circular necrotic spots on foliage in potato varieties, Arran Consul, Arran Banner, Duke of York, Eclipse, Majestic, Up-to-Date, Burbank, Bliss Triumph, President, Irish Cobbler, Earliest-of-All.	Potato Virus C (<i>Solanum Virus 5</i>), p. 359.
" "	In potato Arran Victory, interveinal necrotic blotches on under surfaces of the leaves, followed by wilting and fall of the leaf; later leaves show a pronounced and blotchy interveinal mosaic, together with spotty, black interveinal necroses. Potatoes, President, Arran Chief, British Queen, Edzell Blue, Katahdin, Kerr's Pink, and others show similar symptoms.	President Streak Virus (<i>Solanum Virus 6</i>), p. 362.
" "	Extreme deformity and rugosity of the leaves, together with a blotchy mottling, necrotic spots on the leaf surface and short brown streaks on the veins of the under surface, potato varieties Arran Victory and Arran Chief.	Paraerinkle Virus (<i>Solanum Virus 7</i>), p. 365.
" "	In potato Epicure, general yellowing of the leaves and stems with leaf-drop and premature death; in Dunbar Yeoman, a bright yellow mottling or severe scorching of the lower leaves or both effects in succession, tubers necrosed.	Tuber Blotch Virus (<i>Solanum Virus 8</i>), p. 368.
" "	On Irish Chieftain potato, a brilliant yellow mottle on all leaves of the plant; on President, Early Regent, Majestic, Champion and Ninetyfold, bright yellow spots on the lower leaves, tubers necrotic except those of Early Regent.	Potato Aucuba Mosaic Virus (<i>Solanum Virus 9</i>), p. 372.

Host Plant	Symptoms	Disease caused by
<i>Solanum tuberosum</i> , Potato.	Brilliant yellow or yellowish-white patches on the leaves, plant somewhat stunted.	Potato Calico Virus (<i>Solanum Virus</i> 10), p. 375.
" "	Leaves flaccid and mottled, with an upward rolling, especially distinct in the varieties, Earliest-of-All, Idaho Rural, Bliss Triumph, Green Mountain, and White Rose.	Leaf Rolling Mosaic Virus (<i>Solanum Virus</i> 11), p. 377.
" "	In the Green Mountain variety, shoots more erect, leaves darker green, more rugose; mid-ribs of later leaflets inwardly curled; tubers spindling, long and cylindrical with irregular outlines.	Potato Spindle-tuber Virus (<i>Solanum Virus</i> 12), p. 378.
" "	In Green Mountain potato, dark green foliage, with wrinkling, rugosity and curling; plant dwarfed and spindly, tubers gnarled and cracked.	Unmottled Curly Dwarf Virus ((<i>Solanum Virus</i> 13), p. 379.
" "	In potato President, pallor of young leaves followed by stiffness, slight rolling and upright habit. In most of the other potato varieties, more pronounced rolling of leaves with pigmentation and occasional aerial tubers.	Potato Leaf-roll Virus (<i>Solanum Virus</i> 14), p. 382.
" "	Chlorosis of the leaves with marginal flavescence, production of spindling axillary branches along the stems; long spindling sprouts on tubers which may proliferate and produce chains of small tubers; many little aerial tubers with leafy eyes commonly develop on the main stem.	Potato Witch's Broom Virus (<i>Solanum Virus</i> 15), p. 385.
" "	Plant somewhat dwarfed, stems yellowish-green in colour; death of growing apex; the stem when split shows rusty specks in the pith and cortex of the nodes, tubers small, often cracked, flesh discoloured with rusty brown specks in the pith. Old seed tuber usually unrotted.	Potato Yellow Dwarf Virus (<i>Solanum Virus</i> 16), p. 388.

Host Plant	Symptoms	Disease caused by
<i>Solanum tuberosum</i> Potato.	Rolling of the apical leaves only, chlorosis of the upper part of plant.	Apical Leaf-roll Virus (<i>Solanum Virus 17</i>), p. 389.
" "	Marginal yellowing, upward rolling and cupping of the basal portion of the smaller leaflets; distinct reddish or purplish colour; older primary leaves roll upward over the mid-rib, become yellow and necrotic.	Psyllid Yellows Virus (<i>Solanum Virus 18</i>), p. 391.
" "	Pronounced puckering and downward curling of the leaves; no distinct spotting, but diffused slightly yellowish areas occur all over the foliage. Plants are bushy and dwarfed.	Potato Crinkle (<i>Solanum Viruses 1 and 3</i>), p. 391.
" "	Lower leaves generally with black necrotic veins, upper leaves mottled with light green spots; foliage wrinkled or ruffled, particularly on the Bliss Triumph variety; plants dwarfed and curled with rugose, abnormally hairy leaves.	Potato Rugose Mosaic Virus (<i>Solanum Viruses 1 and 2</i>), p. 394.
<i>Spinacea oleracea</i> , Spinach.	Young leaves show vein-clearing and curling. Later plants turn yellow and die.	Sugar Beet Curly Top Virus (<i>Beta Virus 1</i>), p. 28.
" "	Plant stunted. Young leaves bent downwards, with yellow flecks, which spread and coalesce. Outer leaves necrotic, dying back from tip.	Sugar Beet Mosaic Virus (<i>Beta Virus 2</i>), p. 40.
" "	Young plants show chlorosis which becomes more severe, with malformation, wrinkling and inward rolling of leaves, which may be reduced and feathery. Necrosis develops and spreads inwards from older leaves, finally causing death of plant.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 56.
" "	Plant dwarfed and slightly chlorotic, with many upright secondary shoots. Young leaves show vein-clearing.	Aster Yellows Virus (<i>Callistephus Virus 1</i>), p. 216.
" "	Plant slightly stunted. Leaves yellowed and mottled, sometimes crinkled.	Tobacco Mosaic Virus (<i>Nicotiana Virus 1</i>), p. 236.

Host Plant	Symptoms	Disease caused by
<i>Spinacea oleracea</i> , Spinach.	Plant stunted, yellow. Young leaves show large bright yellow areas, becoming necrotic. No distortion.	Tobacco Ringspot Virus (<i>Nicotiana Virus 12</i>), p. 271.
<i>Taraxacum officinale</i> Weber, Dandelion.	Plant chlorotic with many secondary shoots. Leaves reduced in width, show vein-clearing and reddening or bronzing, upright habit. Flower heads dwarfed.	Aster Yellows Virus (<i>Callistephus Virus 1</i>), p. 220.
<i>Trifolium repens</i> , White Clover.	Leaves mottled with dark green areas adjacent to the main vein, occasionally completely chlorotic.	White Clover Mosaic Virus (<i>Trifolium Virus 1</i>), p. 177.
" "	Chlorotic markings on the leaves, frequently in the form of fine lines or circles. Younger leaves severely dwarfed and deformed with much puckering and twisting of the bud leaves.	Tobacco Ringspot Virus (<i>Nicotiana Virus 12</i>), p. 280.
<i>Triticum sativum</i> , Winter Wheat.	Mottling of irregular streaks on the leaves, dwarfing and excessive proliferation of plant which may be of a rosette form ; leaves later become dark green in colour.	Wheat Rosette or Mosaic Virus (<i>Triticum Virus 1</i>), p. 449.
<i>Tropaeolum majus</i> , Nasturtium.	Older leaves yellow. Secondary shoots formed with dwarfed and malformed leaves. Apical leaves dwarfed with rolled margins. Flower buds dwarfed and chlorotic, calyces and corollas withered or dry.	Sugar Beet Curly Top Virus (<i>Beta Virus 1</i>), p. 28.
" "	Leaves distorted and cupped, irregular pallidity with yellowish spotting, veins sometimes banded with green, numerous necrotic spots later on leaf surface.	Tomato Spotted Wilt Virus (<i>Lycopersicum Virus 3</i>), p. 299.
<i>Tulipa</i> spp., Tulip.	Striping of the leaves in some varieties, with a silvery or light greyish-green colour ; "breaking" of flower colour, consisting of featherings, or stripes and streaks in the petals.	Tulip Break Virus (<i>Tulipa Virus 1</i>), p. 412.

Host Plant	Symptoms	Disease caused by
<i>Vaccinium macrocarpon</i> , Cranberry.	Plants show "witch's broom" appearance. Leaves appear reddish in autumn. Flowers upright; sterile calyx lobes enlarged; petals short, streaked red and green, stamens and pistils abnormal. Flowers may be replaced by whorls of leaves or short branch. Fruits (if any) small, misshapen, held erect; seeds few or absent.	Cranberry False-blossom Virus (<i>Vaccinium Virus 1</i>), p. 205.
<i>Vicia faba</i> , Broad Bean.	Young leaves curled, with blister-like elevations and transparent veins.	Sugar Beet Curly Top Virus (<i>Beta Virus 1</i>), p. 30.
<i>Vicia sativa</i> , Spring Vetch. <i>Vicia aliopurpurea</i> , Purple Vetch.	Youngest leaflets rolled inward along mid-rib, terminal leaflets malformed. Petiole bent downwards, twisted.	Sugar Beet Curly Top Virus (<i>Beta Virus 1</i>), p. 30.
<i>Vicia villosa</i> , Winter Vetch. <i>Vigna sinensis</i> , Cowpea.	Inoculated leaves show dark red necrotic spots. Systemic infection rare, and takes form of yellow lesions and green and yellow mottle, with stunting and distortion.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 64.
<i>Vinca minor</i> , Periwinkle.	Plant slightly stunted. Leaves show severe streaky mottle, with downward curving; internodes short. Flowers reduced in size.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 67.
<i>Viola tricolor</i> , var. <i>hortensis</i> , Giant Trimor-dian Pansy.	Plant stunted. Dense cluster of chlorotic secondary shoots in axils of apical leaves. Leaves rolled inwards. Young leaves show vein-clearing. Veins wavy with small protuberances.	Sugar Beet Curly Top Virus (<i>Beta Virus 1</i>), p. 24.
<i>Viola cornuta</i> , Apricot Queen Viola.	Flower buds often sessile. Flowers dwarfed.	
<i>V. cornuta</i> .	Leaves slightly curled, chlorotic. Flowers with smudged appearance and flecking of petals.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 55.
<i>Vitis vinifera</i> , Vine.	Stocks show poor growth. Leaves with varied mosaic mottle, veins picked out in yellow, or yellow dots or large pale areas	Vine Mosaic Virus (<i>Vitis Virus 1</i>), p. 202.

Host Plant	Symptoms	Disease caused by
<i>Zea mays</i> , Maize.	and bright green patches on leaf; deformation in some varieties. Plants severely stunted. Leaves with light coloured oval spots, crumpled tips, tendency to split. Old leaves with necrotic areas.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 76.
” ”	Small elongated white specks develop on the leaves, later these form fine interrupted stripes which may cover the leaves; breadth of stripes varies according to variety; some phloem necrosis present.	Corn Stripe Virus (<i>Zea Virus 1</i>), p. 439.
” ”	Pronounced chlorosis of the leaves, confined to narrow broken stripes arranged along the veins; chlorotic tissue appears an opaque yellow when viewed by transmitted light. The stripes in the preceding disease (<i>Zea Virus 1</i>) tend to be longer without a break than in the present case.	Maize Streak Virus (<i>Zea Virus 2</i>), p. 444.
” ”	Leaves with a rather diffuse blotching of normal green tissue on a pale green background, the darker areas following the main veins.	Maize Mottle Virus (<i>Zea Virus 3</i>), p. 447.
<i>Zinnia elegans</i> .	Dwarfing, crinkling and rigidity of foliage.	Delphinium Stunt Disease Virus (<i>Delphinium Virus 1</i>), p. 3.
” ”	Internodes shortened. Chlorotic secondary shoots. Flowers dwarfed, petals reduced.	Sugar Beet Curly Top Virus (<i>Beta Virus 1</i>), p. 31.
” ”	Leaves show vein-clearing, followed by severe mottling and distortion. Flowers with distorted, stiff and abnormally coloured petals. Flower stalk short, number of flowers reduced.	Cucumber Mosaic Virus (<i>Cucumis Virus 1</i>), p. 68.
” ”	Plant stunted, chlorotic. Flowers malformed, petals rolled, often tubular.	Californian Aster Yellows Virus (<i>Callistephus Virus 1A</i>), p. 222.

Host Plant	Symptoms	Disease caused by
<i>Zinnia elegans.</i>	Plant stunted, may be rosetted. Leaves curled, distorted ; veins thickened on under surfaces. Flowers dwarfed, of poor colour, partially sterile.	Tobacco Leaf-curl Virus (<i>Nicotiana</i> 'Virus 10), p. 265.
" "	Leaves with mosaic mottling which may consist of numerous flecks of dark green or alternatively of chlorotic rings on a darker background ; growing points sometimes distorted.	Tomato Spotted Wilt Virus (<i>Lycopersicum</i> 'Virus 3), p. 302.

ADDENDA

THE following two plant viruses were inadvertently omitted from the book :—

RUMEX VIRUS 1. Grainger

Synonym. *Dock mosaic virus*, Grainger, 1928.

The Virus

Thermal Death-point. Inactivation occurs at 80° C. after ten minutes' exposure.

Dilution End-point. This is very low, about 1 : 100.

Resistance to Chemicals. The virus is inactivated by 2 per cent formalin in thirty minutes, but not by 95 per cent alcohol in the same time.

Resistance to Ageing. Extracted virus sap is still infectious after fourteen days, and dried leaves after twenty-one days.

Filterability. The virus does not pass a Jenkins filter or an English Berkfeld candle.

Transmission. The virus is transmissible mechanically by tissue mutilation, but not by needle prick. It is not seed-transmitted, and the insect vector is not known.

Disease Caused by Rumex Virus 1

Polygonaceæ

Rumex obtusifolius Dock. *Dock mosaic*. Infected plants are not markedly stunted, nor do they show much variation from the normal. There are areas of a greenish-yellow colour, lighter in colour than the background, on the leaf blades. These areas are sometimes interveinal or they may extend over several veinlets. Symptoms are masked at temperatures of 75° C. and over. The virus is also transmissible to *Rumex lanceolatus* and *R. sanguineus*. (GRAINGER, J., and COCKERHAM, G., 1930 "Some Properties of the Virus Extract of Dock Mosaic." *Proc. Leeds Phil. Soc.*, 2, 406–415.)

ABUTILON VIRUS 1. Baur

Synonym. *Abutilon Infectious Variegation (Virus)*, Baur, 1906.

The Virus and its Transmission. This virus is transmissible by grafting, but not by sap-inoculation; the insect vectors, if such exist, are not known. The virus is sometimes transmitted through the seed of certain hybrids of *Abutilon* spp.

Disease Caused by Abutilon Virus 1

Malvaceæ

Abutilon spp. The attractive bright yellow and green variegation in various species of *Abutilon* is well-known, and is the chief reason for the propagation of the species as an ornamental plant. There appear to be two types or strains of variegation, that occurring in *Abutilon thompsoni*, *A. mulleri* and *A. megapotamicum variegatum* being the more intense form.

Abutilon plants sometimes recover from the disease either wholly or in part, but such plants or parts of plants are susceptible to reinfection. The variegation tends to disappear if the plants are grown in darkness or subdued light. When variegated plants of *A. thompsoni* are kept in total darkness for varying periods of a few days to a fortnight, there is no trace of variegation in the new foliage formed while the plants are in obscurity, and subsequently matured in the light. On the other hand, variegation occurs in the leaves which develop after the plants are restored to the light.

Literature on Abutilon Virus 1

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GENERAL INDEX

Numerals in heavy type refer to descriptions, etc., of the insect vectors.

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